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THE VALUE OF SIALOGRAPHY IN THE DIAGNOSIS OF PAROTID TUMOURS*

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During 1949 a retrospective review of 40 mixed tumours of the major salivary glands revealed that 15% had been misdiagnosed as chronic lymphadenitis or sebaceous cysts.[6] Similar reports from other institutions[1,10] confirmed the need for greater accuracy in the diagnosis of parotid tumours and although there were, at that time, conflicting views about the value of sialography,[2,5] it was decided to investigate its value in the diagnosis of such tumours. Since then sialography has been performed as a routine and 70 sialograms performed on patients with tumours of the parotid salivary glands are reviewed here. This is a selected group of patients because it includes only those cases in whom the exact situation and nature of the tumour have been confirmed by surgical exploration or autopsy, and histological examination.

The pathological nature of these 70 tumours was as follows:

Benign (50): mixed tumour, 34; recurrent mixed tumour, 7; adenolymphoma, 5; oxyphil-cell adenoma, 1; neurofibroma, 1; haemangioma, 1; and lipoma, 1.

Malignant (20): mixed tumour, 2; carcinoma, 14; recurrent carcinoma, 2; and sarcoma, 2.

VALUE OF SIALOGRAPHY

Sialography is based on the concept that a tumour in the salivary gland will interfere with the intra-glandular ducts and that this will be visible on the sialogram. This has been amply confirmed in this investigation and only 1 of these 70 tumours could not be recognized on sialography. This sialogram was performed at the commencement of this series, when I was inexperienced in the technique, but now there is no doubt in my mind that every parotid tumour, sufficiently large to present clinically, can be demonstrated by means of an adequate sialogram. The effect produced by the tumour on the intra-glandular ducts will depend on the type of neoplasm. Broadly speaking we can divide these tumours into 2 big groups: those which are encapsulated and those which invade the surrounding glandular parenchyma. In general the encapsulated tumours are benign and the infiltrating ones are malignant, but this is not always true[3] and consequently it is not always possible to determine the histological nature of a given tumour by means of sialography.

ENCAPSULATED TUMOURS

Encapsulated tumours displace surrounding structures with the result that the intra-glandular ducts will be seen to curve around the tumour without being invaded. These ducts will consequently not be obstructed or damaged and they will be seen in their entirety.

Benign Mixed Salivary Tumours

In the present series, 26 of the 34 clinically benign mixed salivary tumours demonstrated this feature. In 22 cases the duct displacement could be seen on the antero-posterior view, which revealed that the tumour was very superficial in 4 cases (Fig. 1), in the body of the gland in 6 (Fig. 2), and deeply placed in 12 cases (Fig. 3). In most of these the tumour was also visible on the oblique view (Fig. 4), but it is not always seen on this view. In 4 instances the tumour was only recognizable on the oblique picture and not on the antero-posterior view (Figs. 5 and 6); this illustrates the necessity of taking both these views in every case.

All other encapsulated tumours will naturally give similar sialographic appearances and the other 9 primary benign tumours in this series could not, by means of sialography alone, be differentiated from the more common mixed tumours. In addition, other localized, encapsulated lesions will produce a similar picture, and during this investigation 3 branchial cysts,[8] 3 lymphomata and 4 cases with chronic lymphadenitis of the parotid lymph nodes[9] have been seen, which all displaced ducts in this way and simulated benign encapsulated neoplasms.

Superficial Tumours

Tumours on the outskirts of the parotid salivary gland do not displace the intra-glandular ducts sufficiently to be visible on a sialogram and such peripherally placed neoplasms will thus be missed unless special precautions are taken. The very superficial tumour produces a small indentation of the surface of the gland which can only be seen on a special acinar-filling sialogram. This requires a repeat injection of a larger volume of contrast medium to fill the acini of the gland which then becomes visible as a diffuse opaque structure on the sialogram.[2,11-13,15] The tumour appears as a superficially-placed filling defect in this opacity and, in the present series, 6 of the benign mixed salivary tumours could be demonstrated by this method only. In 5 instances the filling defect was apparent on the antero-posterior view (Fig. 7) and in 1 case only on the oblique view.

Other superficially-placed encapsulated lesions will naturally produce a similar filling defect and during this investigation 2 lymphomata and 3 instances of chronic lymphadenitis of the parotid lymph nodes were encountered with identical sialographic appearances.

It is obvious that if a suspected parotid tumour is not

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demonstrated on the more orthodox type of sialogram, a special acinar-filling picture must be obtained to exclude a very superficial neoplasm. It must, however, be kept in mind that such acinar filling obscures the detail of the intra-glandular ducts[2] and may thus disguise ductal displacement due to a neoplasm. For this reason it is important that this method should not be used until the routine ductal pictures have been studied.

Deep Tumours

A tumour on the deep aspect of the gland also does not distort the intra-glandular ducts but pushes the whole gland laterally. Such displacement may be very difficult to recognize, and while the many fallacies and pitfalls of measurements in radiology are appreciated, experience so far indicates that the normal gland and main duct should not be more than 18 mm. and 17 mm. respectively

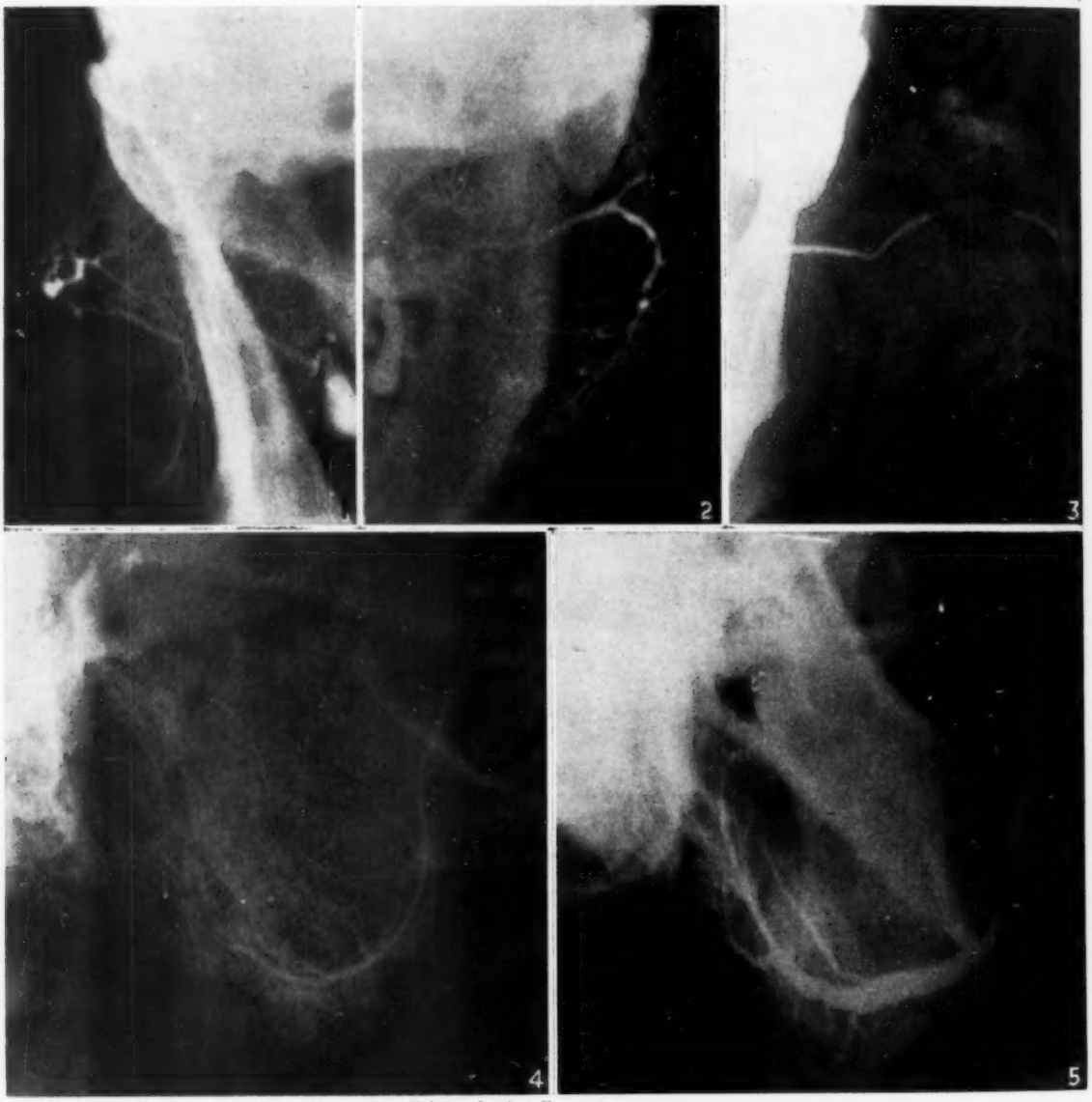

Fig. 1. Displacement of ducts by a superficial mixed salivary tumour.
Fig. 2. Displacement of ducts by a mixed salivary tumour in the middle of the parotid salivary gland.
Fig. 3. Displacement of ducts by a deeply placed mixed salivary tumour.
Fig. 4. Displacement of ducts by a mixed salivary tumour demonstrated on an oblique view.
Fig. 5. Duct displacement by a mixed salivary tumour as seen on the oblique view.

from the lateral edge of the ascending ramus of the mandible on the antero-posterior view. Displacement of either beyond these arbitrary limits is at present considered abnormal and in one instance in the present series this was the only feature which enabled the diagnosis of such a deeply placed mixed salivary tumour to be made (Fig. 8).

Lesions of the deeply placed parotid lymph nodes and of the ascending ramus of the mandible will also displace

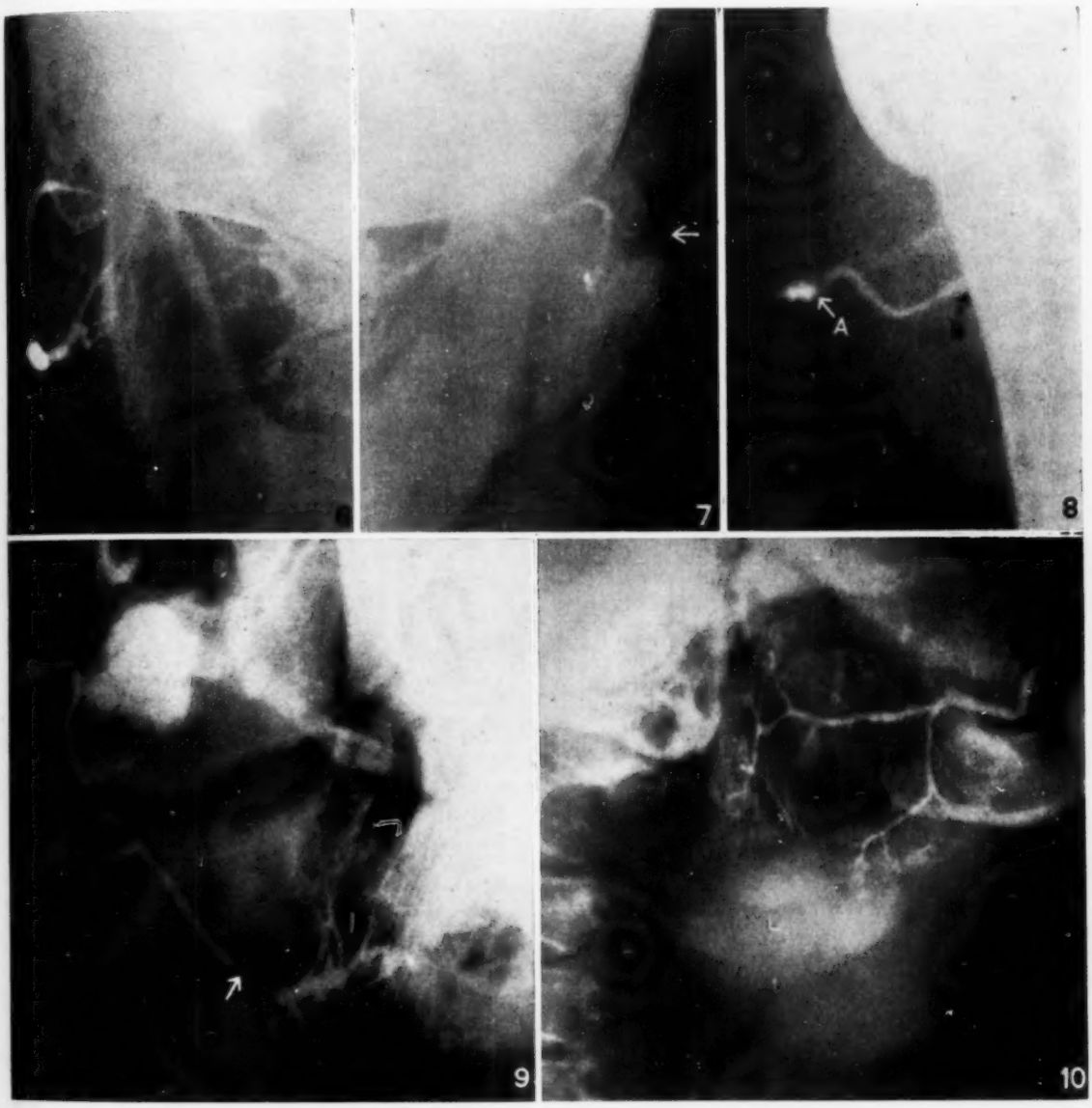

Fig. 6. An A.P. view of the same case as Fig. 5, showing an apparently normal picture.

Fig. 7. Filling defect caused by a very superficially placed mixed salivary tumour as seen on the A.P. view of a special acinar-filling sialogram. (Arrow points to filling defect.)

Fig. 8. A very deeply placed mixed salivary tumour which has pushed the whole gland laterally without obvious intra-glandular ductal displacement. A=main duct, B=lateral edge of ascending ramus of the mandible. Distance between A and B is more than 17 mm.

Fig. 9. Narrowing of an intra-glandular duct with proximal ductal distension produced by a carcinoma. (Arrow indicates narrowing.)

Fig. 10. A carcinoma producing displacement of some ducts but complete obstruction of others.

the parotid salivary gland outwards in this manner. The former conditions cannot be differentiated on the sialogram but disorders of the mandible will, of course, be recognizable on the radiograph, as happened in two instances during this investigation. In the one case there was an osteomyelitis and in the other an osteogenic sarcoma of the mandible.

Encapsulated Malignant Tumours

The sialographic appearance of encapsulation is unfortunately not synonymous with benignity and in this series 10 of the 18 primary malignant tumours (56%) simulated encapsulated tumours on the sialograms. These cases can be divided into 3 groups:

1. Three cases were very slow-growing malignant neoplasms which merely displaced the intra-glandular ducts. They were all slow-growing, well-differentiated carcinomata of 7 years, 3 years and 18 months duration respectively. All 3 tumours were resectable.

2. Five cases were very early carcinomata with an average duration of 3 months. These were still so localized that the neighbouring ducts had not yet been invaded[2,10] and the sialograms showed only duct displacement of the intra-glandular ducts in 4 instances and of the main extra-glandular duct in 1. Four of these 5 tumours were resectable but the other one could not be completely removed.

3. The 2 sarcomata also resembled encapsulated tumours on sialography; this is not surprising in view of the well-known tendency for a soft-tissue sarcoma to form a pseudo-capsule by compression of the surrounding structures. This false capsule is responsible for displacing the intra-glandular ducts to give the sialographic appearance of an encapsulated tumour.

Secondary malignant deposits in the parotid lymph nodes also merely displace the intra-glandular ducts during the early stages, before the malignancy has burst through the lymph-gland capsule to invade the surrounding tissues. During this investigation duct displacement only was found in 3 such cases (2 secondary carcinomata and 1 secondary melanoma) and in 1 other case with a metastatic carcinoma a filling defect in an acinar-filling picture was seen.

INFILTRATING TUMOURS

Once a neoplasm invades the intra-glandular ducts, this will be evident on the sialogram in addition to the displacement of ducts. A confident diagnosis of malignancy can then be made. In this series such evidence was present in 8 of the 18 primary malignant tumours (44%), which is lower than the 62% quoted by Blady and Hocker.[2] The exact effect on the intra-glandular ducts will depend on the degree and duration of the malignant process.

The earliest sign of invasion is an incomplete obstruction of one of the intra-glandular ducts. Not only can the area of narrowing of the duct be seen, but the duct proximal to this obstruction is distended (Fig. 9). In this type of case a refinement in technique which is of additional value is the secretory sialogram.[12,13] After the sialogram has been completed the patient sucks a lemon for 1 minute to stimulate salivary secretion which will wash the contrast material out of the ducts. Five minutes later antero-posterior and oblique views are again taken to assess the extent of the emptying of the gland. In normal glands no contrast material is visible on these 5-minute post-stimulation films. It has been stated[4] that benign tumours produce retention of contrast medium on these special films but this belief has not been confirmed in this series. During this investigation secretory sialography was performed on 28 benign neoplasms in which ductal filling only had been produced with 'neo-hydriol fluid' and in all these cases the ducts were completely empty on the 5-minute post-stimulation films. An additional 6 cases required acinar filling with the same substance and, of these, 4 emptied completely with this technique, but in the remaining 2 cases there was a faint residual cloud of contrast material in the gland.

In this series 3 carcinomata produced incomplete obstruction of an intra-glandular duct with proximal distension and in all 3 cases there was retention of the radio-opaque substance on the 5-minute post-stimulation film. These cases had a varying duration of 3 years, 18 months and 3 months respectively and were all resectable.

Eventually a malignant tumour will destroy one or more ducts completely and this is manifest on the sialogram in two different ways. Either the duct will be completely blocked (Fig. 10), or it may open into a necrotic area which will be seen on the sialogram as extravasation of the radio-opaque material into the tumour (Fig. 11). In this series there were 4 late cases demonstrating these features: 2 were clinically malignant mixed salivary tumours with a duration of 14 and 20 years respectively and the other 2 were carcinomata with a duration of 5 and 2 years respectively. This radiographic appearance was found to indicate an advanced stage of malignancy and of these 4 cases, 2 had distant metastases, 1 had extensive lymphatic invasion and 1 was invading the mandible and the base of the skull.

Once again it is obvious that an advanced malignant metastasis in the parotid lymph glands will also produce a similar effect and during this investigation 1 secondary carcinoma and 1 secondary retinoblastoma were found with duct displacement and complete destruction causing obstruction and extravasation.

In this series there was 1 extremely anaplastic carcinoma with a duration of only 1 month that was so rapidly invasive that it destroyed the intra-glandular ducts without any preliminary displacement — the resultant picture was one of gross duct destruction with extravasation but without any displacement. In spite of the short history there was already significant lymphatic invasion and such a sialographic appearance has a grave prognostic significance. A similar picture was obtained in a patient with an epithelioma of the skin over the parotid gland which invaded the salivary gland by direct spread, but such a condition can be readily diagnosed on ordinary clinical examination.

RECURRENT TUMOURS

Tumours which have recurred following previous operations will naturally produce features similar to those already described, but the operation itself may have damaged the ducts and this may complicate the sialographic picture.

In this series there have been 9 recurrent tumours (2 malignant and 7 benign) and of these 5 produced a sialogram compatible with their pathological natures with-

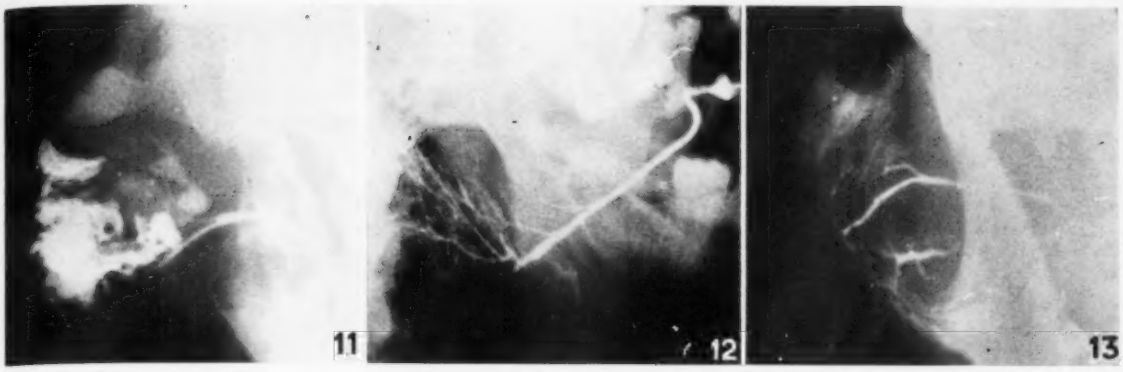

Fig. 11. Extravasation of contrast material into a necrotic carcinoma.
Fig. 12. A recurrent carcinoma producing complete obstruction of a major intra-glandular duct.
Fig. 13. Ductal narrowing and proximal duct distension in a recurrent mixed salivary tumour.

out any evidence of ductal damage resulting from the previous operation. In 1 recurrent adenocarcinoma of 7 years' duration there was destruction and obstruction of the intra-glandular ducts. Of 4 recurrent benign mixed salivary tumours, 3 showed duct displacement seen on the antero-posterior and oblique views and 1 a superficial defect in an acinar-filling sialogram, demonstrated only on the antero-posterior view.

In 4 instances (3 benign and 1 malignant) there was unusual ductal interference, presumably produced by the previous operation. In 2 cases (1 benign and 1 malignant) there was complete obstruction of a major intra-glandular duct which prevented filling of the ducts in a major portion of the gland and consequently no evidence of a tumour could be seen on the sialogram (Fig. 12). In the other 2 recurrences of clinically benign mixed salivary tumours there was displacement of intra-glandular ducts but in addition some stenosis of the ducts with proximal ductal distension (Fig. 13) and delayed emptying on secretory sialograms was seen. This is presumably an effect of invasiveness and it is assumed that it resulted from the damage to the capsule by the previous operation which liberated the tumour into the salivary gland parenchyma as can be seen on histological examination.[7]

DISCUSSION

Sialography can thus be of great assistance to the surgeon who has to deal with a mass in the region of the parotid salivary gland.

It will in every case indicate whether the mass is in the parotid gland or outside it. Every intrinsic mass will be visible on sialography provided the special precautions mentioned in this paper are complied with. Although an extrinsic mass may produce compression or displacement of the gland, the general architectural pattern of the duct system is preserved and there is no localized distortion of the intra-glandular ducts.[8] During this investigation sialography has on many occasions indicated that a mass is outside the parotid gland and a false interpretation in this regard has not been experienced.

A sialogram will also indicate to the surgeon the depth of the mass in the gland. This is of very great assistance to the surgeon during the operation because it will make it easier for him to locate and preserve the facial nerve.

It must, however, be kept in mind that only encapsulation and invasiveness are distinguishable on sialograms and it is impossible to determine with greater accuracy the exact histological type of neoplasm by means of sialography. Generally speaking, benign tumours are encapsulated and malignant tumours are invasive, but this does not always hold good, and it has been shown that 56% of malignant tumours appeared to be encapsulated. If, however, the sialogram reveals some evidence of invasion, then it can be taken as certain that encapsulation is not present. This knowledge is of great value in planning the operation, particularly when the question of preservation of the facial nerve arises.

SUMMARY

A selected group of 70 parotid tumours is reviewed to assess the value of sialography.

It is concluded that sialography will demonstrate an intrinsic neoplasm in every case, provided the investigation is adequately performed.

Various refinements in technique are described which significantly increase the value of sialography.

Sialography can only differentiate between encapsulated lesions and invasive ones and no greater accuracy in diagnosis can be expected.

In this series 44% of primary malignant tumours showed sialographic evidence of invasion. The remainder appeared to be encapsulated but at operation these cases were found to be localized lesions.

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EXTRADURAL HAEMATOMA OF THE POSTERIOR FOSSA

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Extradural haematoma of the posterior fossa is not an uncommon condition. Its presentation is clinically different from the extradural haematoma resulting from a torn middle meningeal artery. It is usually insidious in its presentation and if not recognized early and afforded timely evacuation, it may have a high mortality rate. The following are records of 3 cases encountered in the space of 6 months.

CASE REPORTS

1. J.G., aged 17 years

This patient fell from a horse at 3 o'clock one afternoon. She did not lose consciousness but was dazed. She vomited a few times and complained of a severe headache, and a few hours later became drowsy and was admitted to a hospital under the care of a physician.

Her condition remained unchanged until 3 a.m., twelve hours after the accident. At this time her respiratory rate dropped from 24 to 16 per minute. Her condition rapidly deteriorated and she became comatose, whereupon neurosurgical help was summoned.

In view of the gravity of the situation, only a cursory examination was possible. The patient was in deep coma, responding slightly to painful stimulation, withdrawing the left limbs feebly. Her respirations were deep and sighing at the rate of 6 per minute, and her pulse rate was about 140 per minute. Both pupils were dilated and fixed and she was in a state of complete areflexia.

She was immediately taken to the theatre, but stopped breathing on arrival there. She was intubated and bag breathing was instituted. A right temporal burr hole was made but no extradural haematoma was encountered. A posterior fossa burr hole was then made and a large extradural haematoma was found in relation to a fracture traversing the transverse sinus. Spontaneous respirations resumed shortly after the evacuation. Her recovery was uneventful and she left hospital 2 weeks later with slight residual horizontal nystagmus and left rectus-oculi paresis. She has since made a complete recovery.

2. M.I., aged 35 years

This patient was assaulted with a blunt instrument. He lost consciousness but regained it by the time of admission 2 hours later. His scalp was tightly stretched over a large subgaleal vertical haematoma. His pulse rate was 84 per minute and his respirations 18 per minute. There was marked neck rigidity and the patient was obviously in great pain. There were no lateralizing signs.

X-ray of the skull showed an extensive fracture traversing the sagittal sinus immediately in front of the external occipital protruberance. An incision into the scalp allowed a large subgaleal haematoma to escape. A burr hole placed behind the fracture line revealed a large extradural haematoma, which was evacuated. The bleeding came from the sagittal sinus. The patient's condition remained critical; on the third day he contracted bronchopneumonia and died.

At postmortem examination extensive contusion of the cerebral hemispheres and a small collection of blood in the extradural space of the posterior fossa were seen.

3. M.C., aged 40 years

This patient was admitted to hospital in a semi-stuporose state following a motor-car accident. He complained of severe headache and pain in the nape of his neck. His breathing was regular but at a rate of only 16 per minute, and his pulse was 120 per minute.

He presented with horizontal nystagmus to the left, and slight weakness of the limbs on the left side. All his reflexes were depressed and bilateral Babinski responses were present.

X-ray of his skull demonstrated a fracture in the left parietal region extending posteriorly into the foramen magnum.

At operation, a large extradural haematoma was encountered in the posterior fossa. Bleeding came from the transverse sinus. His recovery was complete and uneventful.

DISCUSSION

Extradural haematoma of the posterior fossa is not uncommon, and it is of paramount importance that it should be suspected when there is marked disproportion between the respiratory and pulse rates. The respiratory rate is characteristically slow and the pulse rate normal or raised. Also, it should be considered when skull X-rays show a fracture traversing the transverse or sagittal sinuses or the confluence of sinuses. The headache and pain in the nape of the neck are severe and characteristically out of proportion to the other signs and symptoms if the patient is conscious.

The signs may vary from a disturbance of respiratory rate only, to cerebellar signs or other signs of involvement of the long spinal tracts. The haematoma is invariably caused by a venous ooze and for that reason will take some hours to develop. This type is more liable to be missed than the extradural haematoma caused by a torn middle meningeal vessel.

When the condition is diagnosed, urgency to evacuate the haematoma is important, and when the condition is desperate, as in case 1, no time should be lost on shaving the scalp or sterilization of instruments.

The haematoma can be evacuated through a suitably placed burr hole, perferably off-centre below the superior nuchal line, but in order to secure adequate haemostasis, a craniectomy of 3 - 4 cm. in diameter should be done.

A lumbar puncture is contra-indicated. Other investigations, e.g. an air encephalogram or angiography, may not be helpful and may be time-consuming.

SUMMARY

1. Three case records of patients with an extradural haematoma of the posterior fossa are presented.
2. The condition may present with a slow respiratory rate associated with a normal or raised pulse rate.
3. Cerebellar signs may be present in the conscious patient.
4. Pain is a prominent symptom.
5. The condition is suspected when a fracture line traverses the larger venous sinuses in the posterior fossa.
6. Speed in its evacuation is imperative in the desperate case.

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EDITORIAL : VAN DIE REDAKSIE

ANTISEPTICS

The problem of local antisepsis is a complex one. A great variety of methods have been used to evaluate antiseptics, but there is still much confusion regarding the best agent to use under various conditions. The traditional phenol-coefficient test, which is thoroughly standardized, has nevertheless proved to be of little practical value because the results obtained bear no relation to the value of the agent on the skin or a wound. In the past too much reliance has been placed on fashion, convention, or convenience, which has handicapped progress. Mercury preparations and other agents have had their day. At present there is a vogue of hexachlorophene preparations and detergents which also require careful assessment.

The number of bacteria on the skin can be reduced, but complete sterilization is impossible, and the surgeon is compelled to operate with hands that cannot be completely disinfected. The skin is not a smooth surface and bacteria are found in large numbers on the surface, in flakes of skin and in hair follicles; and in the skin glands there are transient and resident organisms, which may be virulent or saprophytic in nature. The transient organisms can nearly all be removed by thorough and prolonged scrubbing with brush, soap, and water. The resident population is not so readily removed. In most persons they consist largely of staphylococci of low pathogenicity, but some virulent flora are almost always present, especially when there is frequent and prolonged exposure of the skin to large numbers of pathogenic bacteria. There is a reservoir of deep-lying bacteria as well as superficial flora. There is also an uneven distribution of organisms, but no constant pattern has been determined.

In the disinfection of hands, dirt, grease, and natural fats need to be removed by thorough scrubbing to permit antiseptics to make contact with the cutaneous bacteria. It takes about seven minutes of real scrubbing with brush, soap, and warm water to eradicate all fats from the skin of the hands and arms. No way has been found to prevent an increase of the flora under rubber gloves. Regeneration of the cutaneous bacterial flora, after scrubbing and the use of antiseptics, does occur, and more rapidly under rubber gloves and impervious dressings.

Soap is only a feeble antiseptic, but is of value especially for its detergent or cleaning action combined with washing and mechanical friction. Of the antiseptics that are in use, hexachlorophene is one of the few that does not lose its antibacterial action in the presence of soap. Alkaline soap keeps hexachlorophene in solution, and such combinations have been widely used for preparation of the preparation is 'phisohex'. Hexachlorophene does not, however, disinfect the skin quickly,[1-3] so that preparations conever disinfect the skin quickly,[1-3] so that preparations containing this antiseptic need to be used exclusively and frequently several times a day for four to five days. In this way a reasonably satisfactory 'degerming' of the skin can be anticipated and a film of the agent may be left on the hands and arms. A single brief application would appear to be no more effective than a similar scrub with ordinary soap. Ethyl alcohol, 70 per cent. by weight, is the most effective hand disinfectant available; it must be borne in mind that this preparation does not remain constant in composition in open basins because the alcohol evaporates more rapidly than the water fraction.

The problem of disinfection of the patient's skin differs in several important respects from that of hand disinfection. Space does not permit of any detailed considerations of this subject. With regard to hexachlorophene the same rules apply as indicated above. Patients can be instructed in suitable cases to carry out the regime of repeated washing with the antiseptic at home before an operation. The old-fashioned custom of applying disinfectants for a day or two before surgery, with the part wrapped in sterile dressings or towels, is unsatisfactory.

Further problems exist in connection with the choice of antiseptics for wounds. Here they have very limited usefulness. The chances are that they will be largely inactivated by serum, blood, pus and necrotic tissue, and by dilution, and there are other objections to their use. Finally there are the problems of the topical application of antiseptics in special regions such as the mouth, the urinary tract, and the vagina, and the use of drugs in dermatology.

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BYKOMSTIGE OORWEGINGS BY SPONTANE MISKRAAM

Behalwe die oorwegings in hierdie verband wat ons voorheen al bespreek het,[1,2] is daar nog twee bykomstige faktore waarna ons kortliks wil verwys, naamlik die rol van die saadselle self, en meganiese faktore.

Dit mag wel gebeur dat die ginekoloog te staan kom voor 'n geval van miskraam wat herhaaldelik voorkom (selfs gewoontemiskraam wat per definisie beteken drie opeenvolgende miskrame) in 'n pasiënt wat klinies normaal is volgens alle beskikbare standaarde, en ten spyte van aanvullende hormoon- en dieetbehandeling. Daar bestaan tot nog toe geen finale kennis oor die verband tussen die kwaliteit van die saad (saadsel-morfologie) en die miskraam nie. Deskundiges stem grotendeels saam wat betref defekte in die saadsel-morfologie, en die mening skyn te wees dat baie min sulke saadselle normaal in die servikale slym gevind word — selfs waar 'n hoër presentasie van sulke abnormale selle aanwesig is in die saaduitstorting. Hierdie vorms wys selde goeie beweeglikheid; hulle vermoë om te beweeg is dus besonder beperk sowel wat betref deurdringing van die servikale slym as bereiking van die omgewing van die ovum. Bowendien, selfs saadselle wat normaal voorkom, kan aan genetiese defekte

onderhewig wees en dus aanleiding gee tot miskraam. Daar is op die oomblik geen manier waarop hierdie moontlike defekte vasgestel kan word nie — nie chemies nie en ook nie mikroskopies nie. Die gevolg is dus dat ons slegs maar op 'n verantwoordelike manier oor die saak kan spekuleer in afwagting van verdere werk.

Dat daar meganiese faktore is wat in hierdie toestand van belang is, word vandag algemeen aanvaar. Enigiets wat betreking het op die baarmoederholte, bv. fibroïdes in die baarmoeder of aangebore anomalieë van die baarmoeder, mag wel onvrugbaarheid of miskraam veroorsaak, en moet dus herstel word deur middel van die nodige chirurgiese optrede.

Shirodkar[3] het gedurende die laaste tyd die aandag gevestig op 'n toestand wat bekend is as 'n ‚verswakte servikale mond'. Hierdie toestand word aanvaar as 'n oorsaak van laat miskraam en die chirurgiese metode om dit te herstel is reeds beskryf.

Die serviks bestaan basies uit veselagtige bindweefsel. Van die 14e week van swangerskap af begin die onderste deel van die uterus om te vergroot sodat die produkte van swangerskap, wat nou vinnig groei, bevat kan word. Die vergroting hou skielik op by die aansluiting van die liggaam van die uterus en die serviks. Die serviks word toegehou deur die aard van sy veselagtige struktuur. Wanneer hierdie integriteit belemmer word of verlore gaan, bv. deur vorige geboortes of skeuring of aangebore abnormaliteit, mag die serviks nie in staat wees om die produkte van swangerskap, wat nou al meer en al swaarder word, te behou nie. 'n Min of meer tipiese sindroom mag dus teen die 14e week begin deurdat die vliese skeur met daaropvolgende vinnige geboorte — met of sonder sametrekkings van die uterus.

By die gewone soort miskraam is die volgorde van wat gebeur anders; ook vind dit altyd voor die 14e week van swangerskap plaas. Die ‚verswakte servikale mond'-sindrome word ook gewoonlik voorafgegaan deur 'n geskiedenis van moeilike of voortydse geboorte of met te vroeë afdrukpogings by 'n vorige geleentheid.

Die diagnose word gemaak op die geskiedenis en 'n sterk vermoede dat dit sal gebeur in die volgende swangerskap. Ook moet die serviks gereeld elke week ondersoek word om te kyk of die vliese uitstaan na die 14e week. As die vliese te eniger tyd gesien kan word, is die geval òf een van werklike miskraam wat aan die gang is òf van 'n verswakte serviks, en die nodige maatreëls moet getref word. Sommige skrywers meen dat sodanige verswakking gediagnoseer kan word voor verdere swangerskap plaasvind deur die gebruik van 'n groot verwyder of deur spesiale röntgenstudies, en dat dit chirurgies herstel moet word op hierdie stadium. Die meeste ginekoloë verkies egter om te wag totdat die pasiënt swanger is voordat die finale diagnose gemaak en op behandeling besluit word. 'n Groot mate van sukses is met die laaste metode bereik, en ons moet onthou dat spontane miskraam, wat die gevolg is van aangebore, hormonale of ander oorsake, op hierdie stadium uitgesluit is.

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2. *Idem* (1961): *Ibid.*, **35**.
3. Shirodkar, V. N. (1955): Antiseptic, **52**, 299.

ASSOCIATION OF SURGEONS OF SOUTH AFRICA (M.A.S.A.): SECOND CONGRESS

The Association of Surgeons of South Africa held its Second Congress on 17 - 20 September 1960 in Durban.

Among the 68 delegates attending were two colleagues from Northern Rhodesia and Mr. A. Dickson Wright, F.R.C.S. the retiring Senior Vice-President of the Royal College of Surgeons of England.

The Congress was held at the Medical School, University of Natal, Durban, where all the talks and discussions took place. Clinical demonstrations included cases from the King Edward VIII and Addington Hospitals, and a comprehensive symposium on 'Shark bite', to which Dr. D. Davies of the Oceanarium contributed, was also organized by the Addington Hospital.

Mr. A. Dickson Wright presented two papers during the scientific proceedings and a historical talk during a luncheon on the last day of the Congress. A ceremony was held at the University to admit him to an honorary fellowship of the College of Physicians, Surgeons and Gynaecologists of South Africa.

A popular session was a novel informal luncheon, where groups of surgeons discussed topics of their own choice, e.g. peripheral vascular disease, biliary surgery, pancreatic surgery, breast cancer, neonatal surgery, hiatus hernia, and thyroid diseases.

Throughout the Congress, three pathological demonstrations were on view:

1. Paget's Disease of the nipple (Mr. C. Toker, University of the Witwatersrand).
2. Breast Cancer (University of Natal).
3. Surgical aspects of amoebiasis (University of Natal).

In addition to Mr. Dickson Wright's two papers, 21 other papers were delivered. Of the papers which were presented at the Congress, some are published in full and some in the form of summaries in this issue of the *Journal*.

SOUTH AFRICAN MEDICAL CONGRESS — EXCURSION FARES

Members of the Medical Association, who intend travelling to Cape Town to attend the 43rd Medical Congress on 24 - 30 September 1961, will be interested to learn that the South African Railways offer reduced-rate Excursion Fares *from* all Provinces *to* all Provinces between 15 September and 10 October 1961.

The basis is *ordinary return fare less 25%*, and tickets will be valid to complete the journey within one month of commencement of the forward journey. From 1 April, 1961, also, the holders of four or more return rail-tickets will be able to rail their car at a 50% reduction in charges, e.g. to rail a car form Johannesburg to Cape Town under this scheme will cost R9.50 instead of R19.00.

BREAST CANCER: ASPECTS OF LOCAL RECURRENCE*

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'No matter how early a case of cancer or how thorough the treatment, no patient is free from the possibility of recurrence until death ensues from some other cause.'[1] This fact has been known for many years, and it applies particularly to breast cancer. Why recurrences occur 20 - 30 years after initial operation is debatable, but an interesting observation noted by Sir Gordon Gordon-Taylor[2] is that these recurrences often seem to be preceded by some surgical operation for an independent condition or after a chance intercurrent illness.

By local recurrence is meant the reappearance of cancer, in the area of the operative field or close to it, at any time after the removal of the primary tumour. These recurrences are to be differentiated from regional metastases, so-called 'new' cancers in the other breast, and involvement of supraclavicular or mediastinal lymph nodes. The diagnosis may have to be confirmed by biopsy to differentiate the lesions from 'pseudo recurrences'.[2]

At Groote Schuur Hospital, Cape Town, we saw and treated 67 female patients with recurrent breast cancer during the 6 years, 1954 - 1959. The original treatment of the breast cancer in each case is shown in Table I. During the 6 years 823 patients with breast cancer were seen at the hospital on whom a total of 369 radical and 71 simple mastectomies were performed. In the overwhelming majority of patients the surgical treatment was combined with the application of deep-X-ray therapy.

TABLE I. ORIGINAL TREATMENT OF PATIENTS WITH RECURRENT BREAST CANCER

Treatment	*No.*
Radical mastectomy and postoperative DXR	37
Pre-operative DXR and simple mastectomy	12
Simple mastectomy and postoperative DXR	7
Radical mastectomy only	5
Simple mastectomy only	3
Pre-operative DXR and radical mastectomy	2
Local excision, DXR and androgens	1
Total	67

DXR= Deep-X-ray therapy.

Table II shows the incidence of recurrent cancer as seen at Groote Schuur Hospital. The left-hand column gives the figures for those who were treated by surgery as well as by radiotherapy at the hospital, and compares them with those of patients referred to Groote Schuur Hospital from outlying hospitals for radiotherapy before or after (usually after) surgical ablation, or for hormone therapy. Note that 3 patients who had originally been reported as having tumour-free axillae, and 19 whose axillary lymph nodes had shown signs of invasion, developed recurrences. I shall refer to the 3 patients with no lymph-node involvement again. The higher incidence after simple mastectomy is undoubtedly due to the more advanced nature of the growths in these cases.

* Based on a paper delivered at the Second Congress of the Association of Surgeons of South Africa (M.A.S.A.), Durban, 17 - 20 September 1960.

TABLE II. INCIDENCE OF LOCAL RECURRENCE AT GROOTE SCHUUR HOSPITAL, 1954 - 1959 *

Operation	*GSH patients*	*Referred to GSH for DXR: surgical treatment elsewhere*
Radical mastectomy:		
Operation – no nodes	103	
Recurrences – no nodes	3	
Incidence	2·9%	
Operation – with nodes	107	
Recurrences – with nodes	19	
Incidence	17·7%	
Total:		
Radical mastectomies	210	159
Recurrences	22	22
Incidence	10·5%	13·8%
Simple mastectomy:		
Total	47	24
Recurrences	15	7
Incidence	32%	29·1%
Total number of operations (radical and simple mastectomies)	257	183
Total recurrences	37	29
Incidence	14·4%	15·3%

GSH= Groote Schuur Hospital. DXR= Deep-X-ray therapy.

* One patient who had a local excision of a carcinoma is not included in this table.

TREATMENT AND PROGNOSIS OF PATIENTS WITH RECURRENCES

The recurrences often appeared insignificant: small lumps, coloured red or brown, usually multiple, painless and not tender. Many were frankly ulcerating.

Time of Onset after Original Treatment

Of the patients with local recurrences, 62% developed nodules within 1 year, and 90% within 3 years. This is in accordance with other reports.[3] We have records of recurrences appearing 10 and 12 years after treatment, but such patients do not fall within the scope of this follow-up.

Associated Secondary Manifestations

The incidence of patients developing secondary manifestations in our series, apart from local recurrences, was as follows: nodules in the opposite breast, 22%; skeletal metastases, 24%; intra-abdominal metastases, 11%; and intrathoracic metastases, including lung lesions and pleural effusions, 28%. In 15% of our cases there were only local skin nodules.

The commonest lesions were those found in the thorax, usually in the form of a pleural effusion. Many had skeletal metastases, and the skin nodules usually occurred before the bone secondaries were apparent, but sometimes they were discovered simultaneously. In 15% of the patients the lesions were localized to the original breast area and in a further 22% the nodules had also spread to involve the contralateral chest wall.

Treatment of the Skin Nodules

In many instances the skin nodules were the least serious of the secondary manifestations and treatment was then mainly directed at the other lesions. Where deep-X-ray

therapy had not previously been given, the treatment was often easy and most effective. Radiotherapy then cleared the chest wall in a most gratifying manner. These patients tended to do well, and it is possible that they would not have developed the recurrences had radiotherapy been given at the time of the original surgical treatment.[4]

The treatment of those recurrences which appeared after both surgery and radiotherapy proved extremely difficult. Further radiotherapy often failed to stem the progression. Local excision was feasible on only 2 occasions because of the usual multiplicity of the lesions, and in 1 instance it was followed by the rapid appearance of other nodules all over the chest wall. In no case did we resort to forequarter amputation.[5] In the majority of patients hormonal therapy was given (Table III). There were some encouraging results after oophorectomy in the younger age

TABLE III. HORMONAL TREATMENT OF PATIENTS WITH SKIN NODULES

Treatment	*No.*	*Objective response*	*Subjective response*	*Unchanged*	*Worse*
Oophorectomy	18	6	2	10	—
Testosterone	24	3	4	16	1
Oestrogens	11	—	4	7	—
Ovarian irradiation	1	—	—	1	—
Adrenalectomy	3	—	—	3	—
Hypophysectomy	3	—	—	3	—

group; unfortunately the response was usually short-lived and recrudescence of the lesions became apparent within 12 - 18 months. All these patients are now dead from cancer and in none did the local lesions completely vanish. Testosterone and oestrogen therapy for local recurrences proved disappointing.

Follow-up Studies

Of the patients treated, 11 are alive and free from carcinoma. All had been successfully treated with deep-X-ray therapy, and in 1 the lesion had also been excised. A typical example is Mrs. I. H. (57/02338), 45 years, who had had a simple mastectomy after deep-X-ray therapy for an attached lump, 3 - 4 cm. in size, in the lower and outer quadrant of the left breast, with lymph nodes palpable in

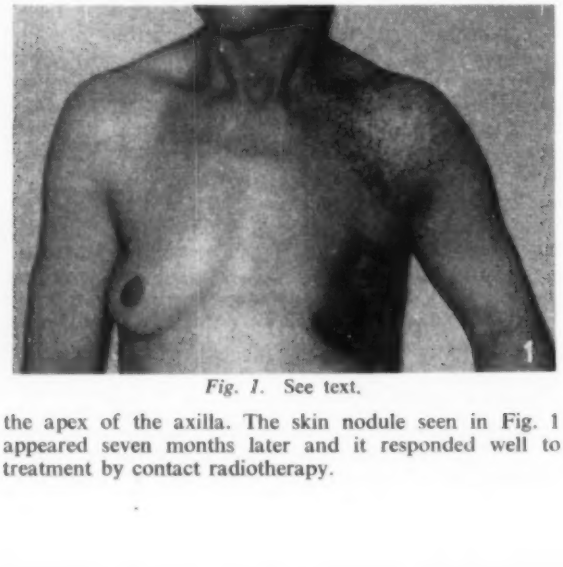

Fig. 1. See text.

the apex of the axilla. The skin nodule seen in Fig. 1 appeared seven months later and it responded well to treatment by contact radiotherapy.

Fig. 2. See text.

Ten patients are alive, but with local disease present and progressing. In 2, the lesions are extending slowly over the years, e.g. Mrs. I. S. (153520), aged 67 years. Local nodules have been present since 1954 and they are extending slowly but surely although she has been given radiotherapy, oestrogens, androgens and 'thio-tepa'. Fig. 2 shows the nodules in 1957, while Fig. 3 shows them in 1959.

The remaining 46 patients have died, still with their local disease, and in many cases with distant secondaries as well. The overall mortality rate in our series is as follows: 1 year after treatment, 40% were dead; 2 years after treatment, 60% were dead; and 3 years after treatment, 72% were dead. This reveals the alarmingly poor prognosis of these patients, a prognosis equivalent to any of the most advanced or 'virulent' forms of cancer. Our findings agree with those of Conway and Neronara,[6] who stated that the onset of local recurrences was a grave prognostic sign and almost all the patients in whom they occurred were dead within 5 years. Some believe that the cancer-free period before the onset of the recurrences may parallel the survival period,[7] but this is denied by others.[8]

It has been stated that the recurrence index is higher in patients treated by simple mastectomy compared with those undergoing radical mastectomy.[9] If this is true, and if the outlook is so bad for those with recurrent breast

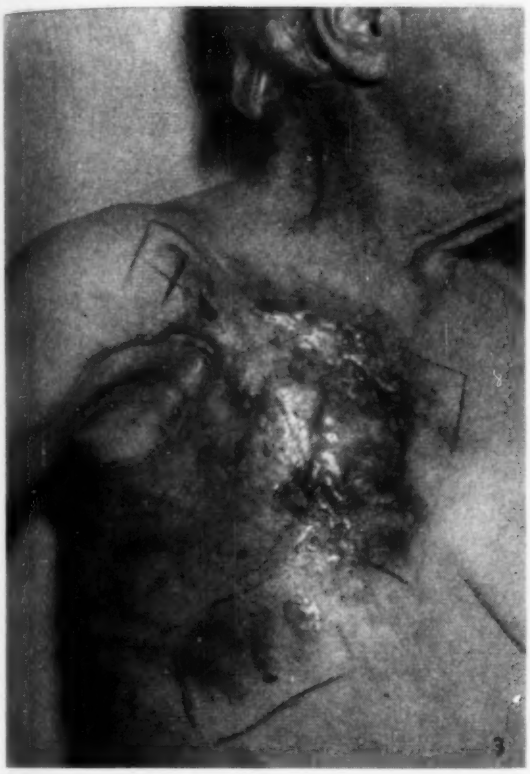

Fig. 3. See text.

cancer, then, surely, a good case can be made for the radical operation.

FACTORS WHICH PLAY A PART IN THE PRODUCTION OF RECURRENCES

In this series the races, age groups and sexes were not factors. During the period under review only 10 male patients were treated, and this accounts for the absence of males in our series.

The Pathology of the Original Lesion

An attempt was made to correlate the histopathological appearance with the clinical progression of these and other patients with breast cancer. This was done by trying to grade them according to the method described by Bloom and Richardson,[10] by noting tubular formation, nuclear pleomorphism, numbers of mitotic figures, and the appearance of hyperchromatic nuclei. Dr. Martin Sacks of the Department of Pathology, University of Cape Town, compared the histology of patients who had recovered exceedingly well after surgical treatment with those who had died or developed extensive recurrences within 1 year of surgical treatment.

The difficulty of grading tumours soon became obvious, and the correlation with clinical progression was impossible in many cases. The appearance of the tumour often varied from slide to slide, and from field to field on the same slide, and it was difficult to know which part of the slide to accept for grading purposes. It was also often difficult to duplicate the grading from day to day and, because of the human factor, cases of moderate malignancy could easily be graded on one day as severely malignant, and on another as mildly malignant. No uniform pathological appearance correlated with clinical follow up, and it seemed reasonable to assume that the pathology, as seen histologically, was probably not an important factor in determining the onset of recurrences.

In 2 of our patients the recurrences may have been, at least, partly due to the nature of the pathology in that they were both cases of sarcoma of the breast. In a 30-year-old African female (A.M., 59/20192), a horrible

Fig. 4. See text.

recurrence grew out only 1 month after local mastectomy (Fig. 4). She died soon after this from her disease, a highly cellular spindle-celled sarcoma.

Influence of Lymph-node or Lymphatic Invasion

This includes intercostal lymph vessels, internal mammary nodes, and axillary lymphatics and nodes, and will be discussed under 4 headings.

1. Axillary Nodal Involvement in Our Series

Clinical and pathological examination revealed axillary node invasion in all save 3 of our patients. However, the subsequent progress in these 3 cases, described hereunder, suggests that they, too, had lymphatic involvement at the time of their original treatment.

(*a*) S. le G. (55/12639). Two years after radical mastectomy she developed a large supraclavicular node on the ipsilateral side, as well as skin recurrences. Four years after operation she had ascites and hepatic metastases. In spite of a negative pathology report, I feel there must have been lymphatic invasion at the time of the original operation to account for the invaded supraclavicular node arising after the mastectomy.

(*b*) C. M. (56/01422), and (*c*) R. M. (138922). Both

these patients developed skin recurrences after radical mastectomy. Both their lesions were in the upper and *inner* quadrants, and in spite of a negative axillary-node report, may well have had internal mammary lymphatic invasion. This seems all the more likely because both developed recurrences in the parasternal area.

2. The Site of the Recurrent Nodules

The nodules tended to congregate around the original scar and in the more advanced cases the whole scar became completely replaced by carcinoma. Closer examination revealed a clustering of the nodules in certain distinct areas, namely in the upper and outer quadrants extending towards the axilla, and also in the parasternal regions (Fig. 5). They seemed to be more numerous in sites where lymphatics were mainly concentrated or possibly more exposed, i.e. in the regions of the axillary and internal mammary lymphatics and lymph nodes. This is well demonstrated in Figs. 6 and 7. Fig. 6 shows the local mastectomy scar of M.M. (55/792) in 1957. Fig. 7 shows the recurrence in 1958, which occurred towards the axilla where lymphatics could be incriminated, and not in very close association with the operation scar. Fig. 8 shows the chest wall of S.M. (57/17228), an example of a recurrence in the parasternal region following a simple mastectomy for a large central growth.

Fig. 5. This demonstrates the numbers of patients in this series with lesions concentrated at the sites shown. For purposes of clarity all lesions are shown as occurring on the right side. In some patients the lesions occurred in more than one area.

It was not uncomon to find recurrences confined to these 2 regions — towards the axilla and in the parasternal area — in one and the same patient. The tendency to congregate in these regions, I feel, lends support to the theory of lymphatic invasion at the time of the original treat-

Fig. 6. 25 July 1957. Local mastectomy for large upper and outer quadrant carcinoma attached to skin and deep fascia. Postoperative radiotherapy given.
Fig. 7. Same patient as in Fig. 6, 16 September 1958. Gross axillary recurrence extending to the front of shoulder. The patient died soon after this.

Fig. 8. See text.

TABLE IV. LOCAL RECURRENCES FOLLOWING RADICAL MASTECTOMY IN VARIOUS SERIES

Recurrences	Roosevelt Hosp.[12]	Henry Ford Hosp.[12]		Johns Hopkins Hosp.[12]		Haagensen[11]	Presbyterian Hosp.[11]	Mayo Clinic* 1945 - 1954	Parsons[10]
	Plastic closure	Plastic closure	Plastic closure + skin graft	Plastic closure	Thiersch graft	Plastic closure + skin graft	Plastic closure + skin graft	Closure of skin	Plastic closure, limited skin excision
	%	%	%	%	%	%	%	%	%
Limited to breast	10·8	5·5	6·1	—	—	4·4	9·7	10	—
Axilla involved	31·5	27	20·8	—	—	24·4	31·3	30	—
All cases	22·7	10·7	15·4	39·7	30·1	11	23	—	8

ment. Occasionally recurrences were seen at sites far removed from the breast area, i.e. over the neck, back, face, and even over the eyelids. We have seen 2 such patients, and in both instances these reacted very favourably, though temporarily, to oophorectomy. There is no reason why lymphatic permeation could not be incriminated, even in these widespread nodules.

3. Influence of Invaded Lymph Nodes on Incidence of Recurrence

It has been shown that the higher the incidence of nodal invasion the higher the incidence of recurrence.[11] The likelihood of recurrences also increases when the lymph nodes are more obviously invaded. This is shown in Table IV, which summarizes recurrence rates as found in the literature, and emphasizes the point that when lymph nodes are frankly invaded, the incidence of recurrence is 3 - 6 times higher.[9, 11-13] Table IV also shows that various types of skin closure bear no relation to the recurrence rate.

Fig. 9. J.G., aged 56 years. She had an upper and outer quadrant carcinoma with dependent *peau d'orange*. Seven months after radical mastectomy the recurrences appeared. This picture was taken 3 years after operation. The patient died soon afterwards from gross local disease.

4. Influence of Oedema of the Breast

Peau d'orange indicates widespread lymphatic permeation, and it is therefore not surprising that some of our worst recurrences occurred after radical mastectomy in those patients who had evidence of this oedema, e.g. J.G. (57/12753), whose extensive local recurrence is well illustrated in Fig. 9. In this series 55% of the patients had oedema of the breast; in some it was frankly dependant and in others there was only a little over the tumour itself. Not only was the incidence of recurrence high in the prescence of *peau d'orange* but, in the cases we saw, the nodules usually arose very soon after operation, sometimes as early as 3 months.

From the 4 points discussed it is reasonable to conclude that lymphatic invasion at the time of the original treatment is the most important single factor that determines the possibility of recurrence.

Cancer Left Behind in the Subcutaneous Tissues or in the Skin Edges

From the surgical point of view the problem of cancer left behind in the subcutaneous tissues or in the skin edges is often mentioned and stressed. This may occur by leaving skin flaps which are too thick, or by not taking away enough skin at the time of the original operation — usually during the radical mastectomy.

1. Leaving Skin Flaps which Are too Thick

Very thin skin flaps are virtual full thickness skin grafts, with an embarrassed blood supply, particularly if there should be any tension. This leads to a high incidence of sloughing, prolonged periods of dressings and further grafting operations. On the other hand thick skin flaps heal much better and sooner, and radiotherapy can be applied earlier, and probably in larger doses.

If one has to depend on the removal of tissue about 1 millimetre thick to cure a cancer patient, then I believe the lesion is already incurable. It is difficult to understand how the removal of a little superficial fascia really can make the difference between cure and recurrence in the operable case. I believe that if a thick skin flap cannot be safely left behind, then the case is incurable. Auchinloss[14] has shown that in spite of meticulous cutting of very thin skin flaps, recurrences do occur too frequently.

2. Leaving Skin Flaps which Are too Large

Table IV shows the influence on the incidence of recurrences of the various types of skin closures, i.e. wide excisions with skin grafts on the one hand, and plastic closures on the other, and it shows that this factor is not a significant one. This is confirmed by Handley,[10] and, interestingly enough, even by Haagensen.[11]

Haagensen, a most meticulous surgeon, who removes skin very widely (at least 7 cm. from the tumour edge

finds that even in his expert hands the recurrence rate of his patients is high. After radical mastectomy, where lymph nodes are invaded, his recurrence rate is no less than 24%. Haagensen blames his criteria for operability; this is surprising, since his criteria for radical mastectomy are extremely strict, and he removes skin very widely – even so, his recurrence rate is still high. At Groote Schuur Hospital we have 3 patients who for various reasons had a simple excision of proved breast carcinoma 2 – 7 years ago and are still without skin recurrences. Their skin excision could not have been more limited.

CONCLUSIONS

Let me hasten to add that I am not advocating any change in the treatment of breast cancer, nor am I advocating less skin removal at the radical mastectomy operation: neither do I suggest limiting the scope of this operation. What I am trying to show is that, when dealing with a clinically operable breast cancer, what matters is the problem of lymphatic invasion. If the lymphatics are not invaded, it probably does not matter whether one takes thick or thin, wide or limited skin flaps — the outcome will always be so much the better. On the other hand, should the lymphatics be invaded, the nodules may well arise in spite of drastic attempts at radical surgery.

It has been stated that 'if persistent disease is shown by local recurrence it must be concluded that the operative procedure was inadequate'.[2] This is correct, but it is also correct to add that the patient should probably not have been operated on at all, since the most extensive surgical procedure possible would not have prevented the manifestations of recurrent disease in such cases.

There are many other factors in the production of recurrences, e.g. the problem of contamination by biopsy and the effect of radiotherapy used to prevent recurrences. These are beyond the scope of this paper.

If lymphatic invasion is so important in this problem, then it becomes all the more important to do the radical operations on early cases. Because selection is always a purely clinical matter, more advanced lesions, unsuspected by the surgeon, will be included in the operable series, accounting for the persistence in the incidence of re-recurrences. Haagensen has stated that to date no way has been found of eradicating this complication.

The results in this series indicate that in the vast majority of cases local recurrence in breast cancer is a sign that lymphatic invasion has taken place, with the consequent probability of widespread metastases apart from the local recurrences. These recurrences, therefore, hold a very grave prognosis.

I feel it appropriate to end by paraphrasing J. Hadfield's[16] quotation of Celsus: There is no so great danger of breast cancer unless it be irritated by the imprudence of the surgeon.

SUMMARY

During the years 1954 – 1959, 67 patients with recurrent breast cancer were seen and treated at Groote Schuur Hospital. The majority of these also had distant metastases. Many of them were temporarily improved by oophorectomy. The mortality rate revealed the serious prognosis of these patients. It was felt that the most important single factor in determining the onset of the recurrences, was lymphatic permeation at the time of the original treatment of the breast cancer. This factor determined the persistence of the disease in spite of attempts at eradication by radical surgery. Included under the heading of radical surgery were such manoeuvres as the cutting of very thin skin flaps and the excision of wide areas of skin around the tumour.

I wish to thank Prof. J. H. Louw for his advice and helpful criticism. I am indebted to Prof. J. Muir Grieve, Dr. M. B. Bennett, and Dr. R. Sealy, of the Department of Radiotherapy, Groote Schuur Hospital, for their advice and for allowing me access to the radiotherapy records of these patients. I also wish to thank Dr. Martin Sacks for his opinion regarding the pathology specimens, and for the great patience he displayed. Dr. J. G. Burger, Medical Superintendent of Groote Schuur Hospital, is thanked for permission to publish these records. My thanks are also due to Miss B. Smit and Mrs. P. Kottler of the Department of Surgery; Miss Smit for assisting with the follow-up of the patients and Mrs. Kottler for preparing the tables. Mr. G. McManus and Mr. B. Todt are thanked for the photographs.

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OPERATIVE CHOLANGIOGRAPHY*

C. A. R. SCHULENBURG, M.D., M.Ch., F.R.C.S., *Pretoria*

Operative cholangiography was first described by Mirizzi[10] in 1932. The present status of operative cholangiography is typically expressed by the following divergent views, as found on successive pages in the *Archives of Surgery*, 1956: M. D. Sachs,[11] of Cleveland, states 'There is universal agreement as to the value of postoperative cholangiography. The equivalent results are obtainable with operative cholangiography. There should be no question as to the value of the procedure'. On the next page, Arthur Allen,[1] of Boston, says: 'It is apparent that the routine use of operative cholangiography is a cumbersome and impracticable solution to the problem of diagnosis and treatment of stones in the biliary ducts'.

I drew attention to the use of operative cholangiography in South Africa in 1956,[18] and in 1957[18] published my observations on 140 consecutive cases of biliary surgery

* Paper presented at the Second Congress of the Association of Surgeons of South Africa (M.A.S.A.), Durban, 17 - 20 September 1960.

in which routine use was made of cholangiography at operation.

Up to 31 January 1960, a further 213 biliary tract operations with operative cholangiography were performed, making a total of 353 for review since I commenced this procedure in October 1955. Although it is not infallible, we have shown that this is a valuable addition to our standard techniques of biliary surgery. It is however, only an *additional* procedure, and under no circumstances should the accepted clinical indications for exploration of the common bile duct be ignored.

Technique

It is obvious that X-ray plates of the highest quality should be obtained if we want reliable information. Therefore, the technique of introducing the dye must be meticulous, the standard of radiography should be excellent, and a radiologist should be present at the operation.

STEPS IN THE PROCEDURE

1. *Tunnel and Casettes*

Arrangements are made for the insertion of the casettes containing the X-ray plates. The most satisfactory is the apparatus devised by Eric Samuel[14-16] (Fig. 1). This consists of a full-length wooden tunnel, which is leadlined except for a space corresponding to the position of the upper abdomen. This space is closed in by a perspex plate with a stationary Potter-Bucky grid built in. The casettes are contained in pockets in a canvas 'bag' which can be extracted to the required spot by a helper at the head of the table. In this way a series of 3 - 5 plates may be taken without disturbing the operating team.

If this apparatus is not available, a suitable tunnel may be constructed of wood, and a casette, together with grid, may be slipped in and out from the sides (Fig 2). Modern operating tables have a suitable tunnel built in.

2. *Position of Patient*

The radiographer checks that the right subcostal area over the midclavicular line corresponds to the centre of the X-ray casette.

The patient is now rotated slightly to the right by placing sandbags behind his left buttock and left lower ribs. This is essential in order to project the common duct clear of the spine. Note that the *patient* should be rotated, not the table; tilting the table will interfere with the vertical projection of the X-rays through the Bucky grid, leading to linear disturbing shadows on the plate.

3. *Laparotomy*

Laparotomy is performed, all abdominal and pelvic organs examined, and biliary pathology confirmed.

4. *Dissection*

This begins at the gallbladder neck, and the cystic duct is positively identified. The cystic artery is tied and divided at this stage if it can be identified.

Two silk ligatures are passed around the cystic duct. The one nearer the gallbladder is tied and held long. The other is kept loose. A lateral opening is cut into the cystic duct between the ligatures. (Note: If there is any difficulty in identifying the cystic duct, cholecystectomy proceeds from the fundus, and only when the cystic duct is finally identified, is cholangiography carried out.)

Meanwhile 35% 'pyelosil' has been drawn into a syringe, and an 8-inch length of No. 2 polythene tubing is attached to the syringe with a well-fitting needle. Airbubbles are excluded, the end of the polythene tube is threaded into the cystic duct for only 1 cm. and the silk ligature is firmly tied over it.

5. *Injection of Dye*

The X-ray apparatus is wheeled into position and serial injections and exposures are now made. When the common duct is not widened, the amounts used are small, 1 ml., 2 ml. and 5 ml. If the common duct appears dilated, we use 2 ml., 5 ml. and 10 ml. Before each injection a new casette is slipped into position.

6. *Cholecystectomy*

This operation is now completed, and appendicectomy and other procedures may also be performed at this stage.

7. *Decision on Duct Exploration*

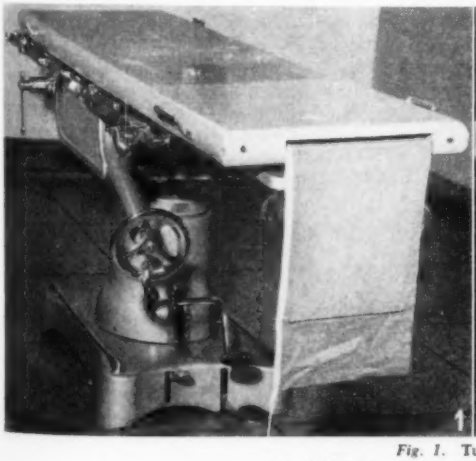
The common duct and the head of the pancreas are now inspected and palpated, and a clinical decision is made regarding the necessity for duct exploration. The X-ray plates are now back for inspection and a final decision is made.

8. *Points to Examine on the X-ray Plate*

(*a*) Width of common duct, (*b*) length and abnormalities of direction of cystic duct, (*c*) exact site of entry of cystic duct into hepatic duct, (*d*) low junction of left and right hepatic ducts, (*e*) accessory hepatic duct, (*f*) stones in common duct, (*g*) other filling defects, (*h*) stricture of common duct, (*i*) flow of dye into duodenum, and (*j*) filling of main pancreatic duct.

9. *Cystic Duct*

Only now, when the anatomy of the biliary tree has been positively demonstrated, is the remnant of the cystic duct dissected out and tied close to the common duct.



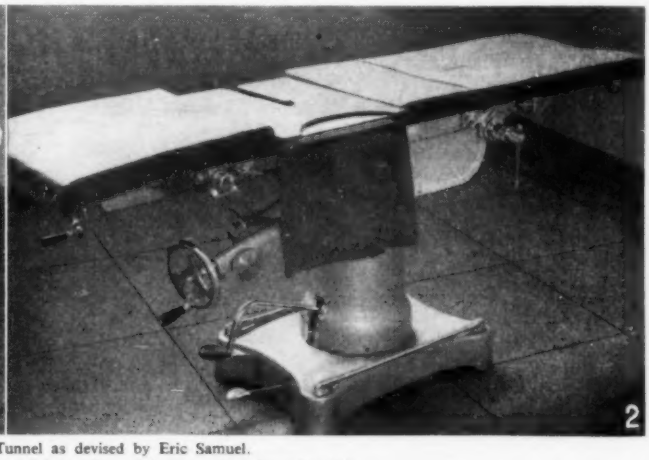


Fig. 1. Tunnel as devised by Eric Samuel.

Fig. 2. Simple wooden tunnel for insertion of X-ray casettes from the side.

10. *Common-duct Exploration*

If an indication for exploration exists, the common duct is opened, stones are removed, the ducts are irrigated, the ampulla is dilated with Bakes' dilators, and choledochoscopy is performed.

A No. 16 Bardex T-tube is cut so that the transverse portion is about 2·5 cm. long. It is cut in the form of a gutter, inserted into the common duct, and sewn in, so that it is watertight, with 00 catgut (plain) sutures. The T-tube should be inserted without trapping airbubbles. To achieve this the tube is filled with saline and clamped. The common duct is also filled with saline. The area around the opening in the duct is flooded, and the T-tube is now inserted 'under saline'. In this manner, airbubbles are eliminated completely from the tube and duct system.

11. *Control Cholangiogram*

A further series of 3 plates is taken as before to check on residual stones, before completing the operation. (Note: Frequently no dye will flow past the sphincter of Oddi at this stage even though a 10 mm. Bakes' dilator had been passed. This is due to spasm and should not cause concern.)

12. *Completion of Operation*

Closure of abdomen, with insertion of Penrose drain, completes the operation.

13. *Postoperative Cholangiogram*

If a T-tube has been inserted, the vertical limb is clamped from the 8th day, and a cholangiogram is done under fluoroscopic control on the 10th - 12th day.

VARIATIONS IN TECHNIQUE

1. *Cholecysto-cholangiography* may be done through a Foley catheter in the gallbladder, or dye is injected directly into the gallbladder by needle in some cases, e.g. in suspected carcinoma of the head of the pancreas.

2. *Direct injection into the common duct* is performed by syringe and needle in cases where the gallbladder had been removed, in cases where the ductal anatomy cannot be identified, and in cases of common duct stricture.

3. *Retrograde removal* of the gallbladder (from the fundus down) may often be necessary before the cystic duct can be identified for cannulation.

4. When stones in the common bile duct are palpable, the operative cholangiogram may be dispensed with, but a *control cholangiogram* at the end of the operation is essential.

5. *Trans-hepatic injection* into a bile duct through a long needle is useful in cases of common-duct stricture.

DANGERS AND COMPLICATIONS

The following have been described, although we have encountered none of them:

1. Pancreatitis.
2. Cholangitis or liver damage.
3. Iodine sensitivity.
4. Bile leakage from needle puncture (drainage should always be provided).
5. Radiation hazards to personnel. We have made measurements and have established that if all personnel stand back 5 feet from the X-ray tube during exposure, they are in no danger.

RESULTS

The results of the operative cholangiography carried out on the series of 353 patients are given in tabular form in this section.

Table I analyses the whole series (353), while Tables II, III and IV deal only with the 326 cases of 'benign biliary-tract disease', the 2 failures mentioned in Table I having been excluded.

Table II indicates that in an appreciable number of cases stones might have been missed but for operative cholangiograms.

TABLE I. OPERATIVE CHOLANGIOGRAMS

Condition	*No.*	
Benign biliary-tract disease:		
Primary gall-bladder disease	295	
Post-cholecystectomy syndrome	23	328
Benign stricture	10	
Carcinoma		17
Miscellaneous		8
Total		353

Of the 353 cholangiograms, 253 were normal, 98 showed abnormalities and 2 were failures. The pancreatic duct was shown in 35 cases (9·9%).

TABLE II. OPERATIVE CHOLANGIOGRAMS IN BENIGN BILIARY-TRACT DISEASE (326 CASES)

	Cases	%
Stones not palpable, but shown on X-ray	23	7
No stones palpable, no history of jaundice, common bile duct not dilated, but stones shown on X-ray	7	2
No history of jaundice, stones shown on X-ray	19	6
History of jaundice, but common duct not explored	22	7

Table III is self-explanatory, showing the low incidence of error in this series.

TABLE III. LOW INCIDENCE OF ERROR IN OPERATIVE CHOLANGIOGRAMS IN BENIGN BILIARY-TRACT DISEASE (326 CASES)

	Cases	
Common ducts explored	113	
Stones shown on X-ray	71	8 false negatives (2·5%)
Stones found in common bile duct	79	
Shadow on X-ray, no stones in common bile duct	2	2 false positives (0·6%)
Residual stones on control (terminal) X-ray	0	
Residual stones on postoperative cholangiogram	2	(in 104 explorations — 1·9%)

Table IV gives a comparison of unselected reports from the literature of the incidence of residual stones, i.e. stones which were overlooked at the times of the choledochotomy. The low figure in the present series is to be noted.

TABLE IV. RESIDUAL STONES AFTER CHOLEDOCHOTOMY ACCORDING TO VARIOUS AUTHORS

	%		%
Hughes *et al.*[6]	24	Buxton *et al.*[2]	9
Corff *et al.*[3]	18	Johnston *et al.*[8]	8
Hicken *et al.*[7]	12	Sullens *et al.*[12]	7·7
Thomson[10]	11	Hight *et al.*[5]	5
Smith *et al.*[14]	10·7	McKittrick *et al.*[9]	2
Glenn[4]	9·7	Present series	1·9

Table V gives the incidence of ductal anomalies encountered in the present series.

TABLE V. ANATOMICAL ABNORMALITIES FOUND IN 351 OPERATIVE CHOLANGIOGRAMS

Abnormal ducts	*Cases*	%
Cystic duct	24	6·8
Hepatic ducts and common bile duct	4	1·1
Accesory hepatic ducts	7	1·9
Total	35	9·8

DISCUSSION

Objections

The following objections to the procedure have been raised:

1. *It is unnecessary*. Some surgeons state that stones in the common bile duct can always be palpated. This is not true. We have been able to demonstrate on 23 occasions that a stone, which was not palpated, could be shown on an X-ray plate, frequently in the presence of an undilated common duct. Some authorities state that they can dissect the anatomy of the biliary tree without difficulty. This is not true, and to attempt it is dangerous. An operative cholangiogram gives us a perfect picture of any anatomical variation in the ductal anatomy, and with this picture before us subsequent dissection becomes quite safe (Fig. 3).

Fig. 3. Accessory hepatic duct (*a*), and stone in common duct (*b*).

2. *It wastes time*. This also is not true. We estimate that the whole procedure takes about 3 - 5 minutes. The operation proceeds in the usual manner while the plates are being processed.

3. *There is fumbling under sterile towels*. This is eliminated by the use of Eric Samuel's tunnel.[14-16]

4. *It is dangerous*. We have encountered no complications following the use of this procedure. Radiation danger to personnel is eliminated if all of them stand back 5 feet from the X-ray tube. At this distance a person will receive a dose of about 1mr. per exposure. The safety margin of radiation is 100 mr. per week.

5. *False positives may occur*. Airbubbles in the biliary tract may simulate stones. This condition will be eliminated simply by ensuring that no airbubbles are introduced. Other filling defects in the ducts may be due to hepatic artery compression, a kink in the common bile duct, overlying gas, etc. A radiologist will be able to diagnose these. In this series there were 2 false positives in 326 cases (0·6%) — see Table III.

6. *False negatives may occur*. This is a real criticism. Dye may surround and totally obscure a small stone. We have practically eliminated this trouble by using a more dilute dye, by taking a series of 3 or 4 plates, by injecting small amounts, e.g. 1 ml., 2 ml. and 5 ml. in a series of 3 exposures, and by insisting on expert radiographic technique (Fig. 4). In this series there were 8 false negatives in 326 cases (2·5%) — see Table III.

7. *Stones may be left after choledochotomy*. We insist on a control cholangiogram by injecting dye through the T-tube at the end of the operation. Care should be taken not to introduce airbubbles. This is done by filling the bile duct and T-tube with saline and inserting the T-tube under a water seal. Numerous reports mention stones discovered in this way in 2 - 24% of cases.[2-9,17-19] In the present series 2 out of 104 cases of duct explorations showed residual stone (1·9%) — see Table IV.

8. *Operative cholangiography might be done only if required, not as a routine*. We cannot see how an accurate selection of cases for operative cholangiography can be made. We have repeatedly demonstrated stones in cases where there were none of the clinical indications for common-duct exploration. We have established that, although not infallible, operative cholangiography can be of very great help indeed. With films of excellent quality and the help of an experienced radiologist in interpretation, a high degree of accuracy can be obtained. I have used the procedure as a routine in 353 cases up to 31 January 1960, and consider that it should be so used. I believe that attempts to use it only in 'selected' cases are wrong, and that much pathology will be missed.

Duct Anomalies

It is noteworthy that in all papers on operative cholangiography, attention is paid almost exclusively to the question of stones in the common duct.

A plea is made here for more attention to anomalies of the cystic, hepatic and common bile ducts. We have found a duct anomaly in 35 out of 351 cases (9·8%) — see Table V. It has been of the greatest help to demonstrate the presence of an accessory hepatic duct, a low junction of the left and right hepatic ducts, the cystic duct entering an accessory hepatic duct, the cystic duct entering the right hepatic duct, the cystic duct entering to the left of the common duct, and the ampulla of Vater situated in the third part of the duodenum (Figs. 5 - 7).

With the anatomy of the biliary tree perfectly outlined on the X-ray plate, it has been possible to plan dissection accurately and to avoid injury to anomalous or normal ducts.

Common-duct Stricture

If dye can be injected above the stricture, the X-ray photograph shows the exact location of the narrowing, the presence of stones above, and frequently the condition of the duct below (Fig. 8).

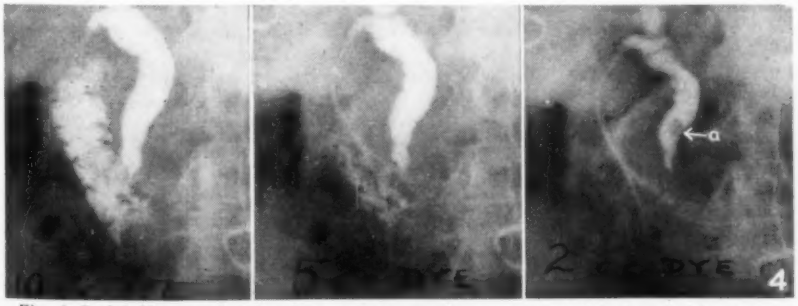


Fig. 4. Serial X-rays, showing how a common-duct stone may be obscured by overfilling. Stone (*a*) seen only on the '2 c.c.' plate.

Carcinoma of the Pancreas

Usually there is no difficulty in identifying the blunt obstruction of a malignant lesion, and it is a great help when a decision has to be made concerning pancreatoduodenal resection (Fig. 9).

Post-cholecystectomy Syndrome

Dye injected directly into the common duct, the gall-

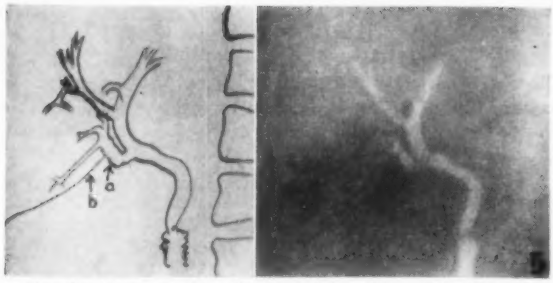


Fig. 5. Accessory hepatic duct (*a*), with cystic duct (*b*) entering it.

Fig. 6. Low junction of left and right hepatic ducts (*a*).

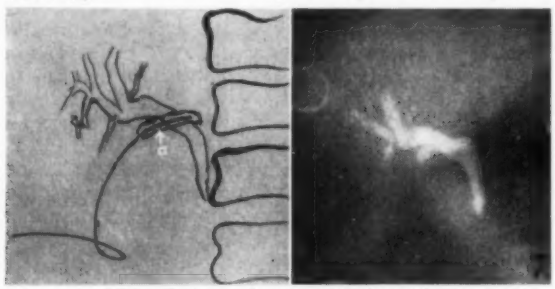


Fig. 7. Anomaly of cystic duct (*a*).

bladder having been previously removed, is usually of great help in showing residual or recurrent common-duct stones, a cystic-duct stump with or without a stone, a gallbladder remnant, common-duct strictures, and stenosis of the sphincter of Oddi (Fig. 10).

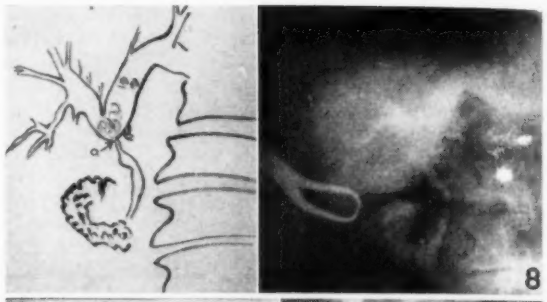
CONCLUSION

It is seen, therefore, that operative cholangiography is helpful in identifying stones and obstructions, and is also of the greatest importance in providing an accurate picture of the anatomy of the bile ducts and in directing the surgeon's attention to abnormal anatomy.

THE FUTURE

Having established the fact that operative cholangiography is not only useful but essential during biliary tract operations, it remains for us to improve the present technique.

1. We should insist on expert radiographic and radiological help.
2. A built-in overhead X-ray tube, and a specially constructed table with a tunnel and movable Bucky grid, may give better X-ray photographs.
3. An image intensifier may be used.
4. Television X-ray: With this technique, a television screen in the theatre visible to the operator demonstrates the radiographic picture of the common duct continuously while the dye is being injected. Viewing takes place under daylight conditions. Although it is outside the scope of this paper, it is suggested that a choledochoscope be used whenever the common duct is opened.



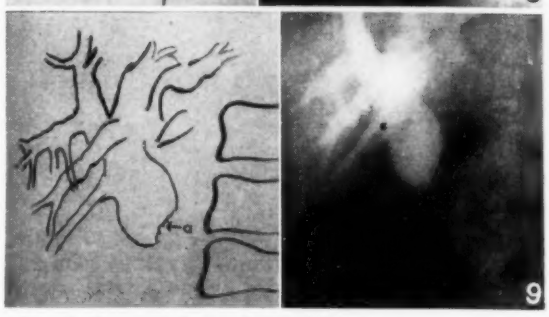


Fig. 8. Common-duct stricture (*a*).
Fig. 9. Carcinoma of head of pancreas, showing blunt obstruction (*a*) of common bile duct.

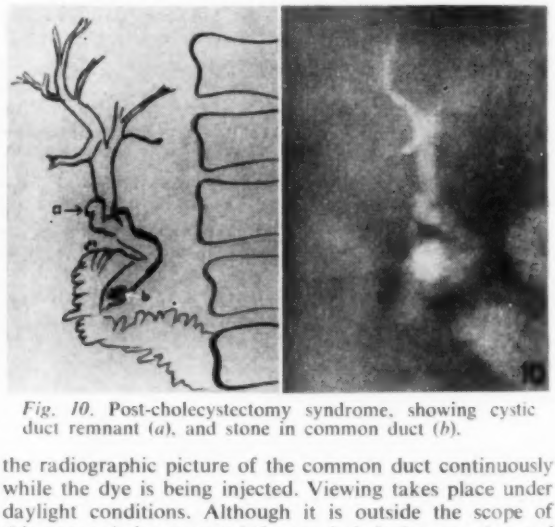


Fig. 10. Post-cholecystectomy syndrome, showing cystic duct remnant (*a*), and stone in common duct (*b*).

SUMMARY

1. A detailed analysis of 353 biliary operations, where operative cholangiography was used, is given.
2. The technique of the procedure is described, and stress is laid on the necessity for meticulous attention to detail in the injection of the dye, as well as on the need for the highest standard of radiography.
3. Complications and criticisms of the procedure are

discussed. It is shown how false positive and false negative readings have almost been eliminated by improvements in technique. A low incidence of residual common-duct stones is reported.

4. Stress is laid on the use of cholangiography at operations to demonstrate anatomical anomalies such as an accessory hepatic duct, a low junction of the hepatic ducts, anomalies of the cystic duct, etc. Operative trauma to such ducts is then easily avoided.

OPSOMMING

1. 353 persoonlike gevalle van operatiewe cholangiografie word ontleed en resultate word bespreek.

2. Die tegniek van die prosedure word breedvoerig beskryf. Klem word gelê op die belangrike punte van die tegniek, en 'n hoë standaard van röntgenologie word vereis.

3. Die nadele van operatiewe cholangiografie word bespreek. Deur verbeteringe in die tegniek het dit moontlik geword om vals negatiewe en vals positiewe bevindings feitlik uit te skakel. 'n Baie lae voorkoms van agtergeblewe choledochus-stene word gerapporteer.

4. Operatiewe cholangiografie is belangrik om abnormale anatomie aan te toon soos bv. bykomstige lewerbuise, afwykings van die ductus cysticus, ens. Operatiewe skade aan die buise word dus vermy.

I should like to thank Drs. A. v. B. Smith, F. W. McLachlan and A. C. McDonald, radiologists, for their help and advice in this work.

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ANALGESIA IN BILIARY PAIN*

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Biliary colic is so severe that women describe it as being worse than childbirth. In the treatment of this pain morphine is said to be contra-indicated. This is based on the thesis that morphine causes spasm of the sphincter of Oddi[1-6] and of the gallbladder.[7-8]

There is no doubt that the pain produced by a gallstone in the common bile duct may be aggravated by morphine,[8,9] and in fact morphine can produce biliary colic in the absence of gallstones,[8,9] presumably by its effect on the sphincter of Oddi. However, many older clinicians[10-12] maintain that morphine is the drug of choice in gallbladder pain.

My interest in this subject was aroused by a well-known clinical observation.[13] Patients often present with a history of repeated attacks of gallbladder pain due to impaction of a gallstone in the neck of the gallbladder. These attacks start suddenly and the pain is relieved immediately by an injection of morphine. There is no recurrence of the pain after the effect of the morphine has worn off. Four such patients admitted to our wards during the past 2 years were carefully questioned, and confirmed this observation.

The relief may be due to the central analgesic effect of the morphine,[1,5,14] but this does not explain the sudden cessation of the pain, with no aftermath or recurrence when the effect of the morphine has worn off.

It also appears to be unphysiological for morphine to produce spasm of the sphincter and contraction of the gallbladder simultaneously.[15,16] Therefore there may be another explanation for the sudden relief of pain in these cases, namely, disimpaction of the gallstone from the neck of the gallbladder – possibly due to the effect of the morphine on the biliary system.

* Paper presented at the Second Congress of the Association of Surgeons of South Africa (M.A.S.A.), Durban, 17 - 20 September 1960.

The purpose of this investigation was to find a suitable drug to relieve biliary pain by determining the actions of various analgesics and antispasmodics on the sphincter of Oddi and the gallbladder.

PRELIMINARY INVESTIGATIONS

Preliminary investigations, which dealt with the effects of drugs on the sphincter of Oddi, have already been published[17] and will be referred to only briefly in this article. They were carried out as follows:

Material and Methods

Patients who had undergone exploration of the common bile duct with T-tube drainage were investigated. In all of them the short limb of the T-tube was at least ½ an inch away from the ampulla of Vater. The sphincter of Oddi was intact and normal in each of the patients tested.

The pressure apparatus (Fig. 1) consisted of a water manometer which was connected to the T-tube and by means of

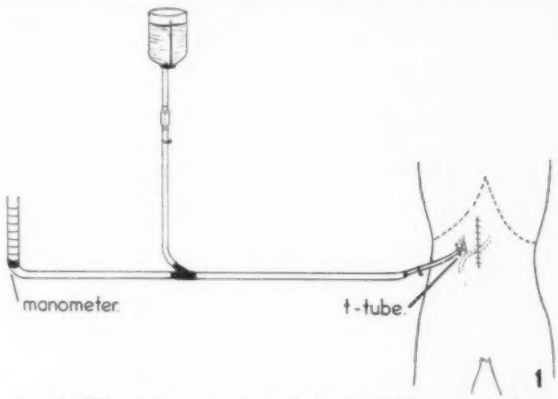

Fig. 1. The simple apparatus used for determining common-duct pressures.

a Y-connection to a vacolitre of normal saline which was used to fill the system. The whole system was filled with saline and the drip was then clamped off.

Once the level in the manometer was constant the patient was given amyl nitrite to inhale. This relaxed the sphincter of Oddi completely and the reading on the manometer was taken as zero.

Effect of Various Analgesics

The graphs in Fig. 2 show the effect of 3 analgesics on the sphincter of Oddi. The resting pressure in graph 2A was 15 mm. of water. After inhalation of amyl nitrite the tone of the sphincter of Oddi was released and the pressure fell to zero.

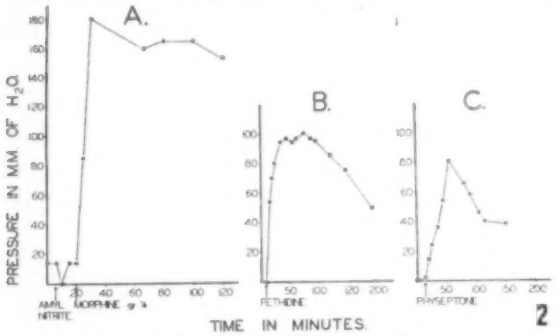

Fig. 2. Graphs showing the effects of (A) morphine, (B) pethidine and (C) physeptone on the intrabiliary pressure.

After the short-lived effect of the amyl nitrite had worn off the sphincter tone returned. Morphine, ½ gr., was given intramuscularly and within 5 minutes the pressure started to rise, reaching a peak of 180 mm. of water after 15 minutes. This rise in pressure lasted about 2 hours. A similar rise in pressure was produced by pethidine and 'physeptone' (Graphs 2B and 2C).

Of all the available analgesics tested in this way (Table I) only 'avafortan' did not produce spasm of the sphincter of Oddi.

TABLE I. THE EFFECTS OF VARIOUS ANALGESICS ON THE SPHINCTER OF ODDI

Drug	*No. of administrations*	*Degree of spasm*			
		Nil	*Min.*	*Mod.*	*Marked*
Morphine	36	0	0	3	33
Omnopon	2	0	0	1	1
Pethidine	5	0	0	3	2
Physeptone	2	0	0	2	0
Avafortan	9	9	0	0	0
Baralgin	2	0	2	0	0

Min. = minimal, Mod. = moderate.

Combinations of various analgesics and antispasmodics were tested in a similar way.

The graphs in Fig. 3 show the effect of morphine combined with various antispasmodics on the intrabiliary pressure. Amyl nitrite produces immediate and complete relaxation of the spasm of the sphincter of Oddi caused by morphine (Fig. 3A). Unfortunately amyl nitrite cannot be inhaled for a long period, therefore its effect is short-lived and the pressure rises soon after the inhalation is stopped.

The combination of morphine and atropine is recommended by many[10-12] as the ideal analgesic in biliary pain on the assumption that atropine acts as an antispasmodic.[5,13] As can be seen in Fig. 3B, atropine does not prevent the marked spasm and increase in intrabiliary pressure produced by morphine. The use of atropine as a biliary antispasmodic therefore appears ill-founded.[19,20]

Intramuscular aminophylline (Fig. 3C) maintains partial relaxation of the sphincter of Oddi, after an injection of morphine, for approximately an hour. Thereafter the intra-

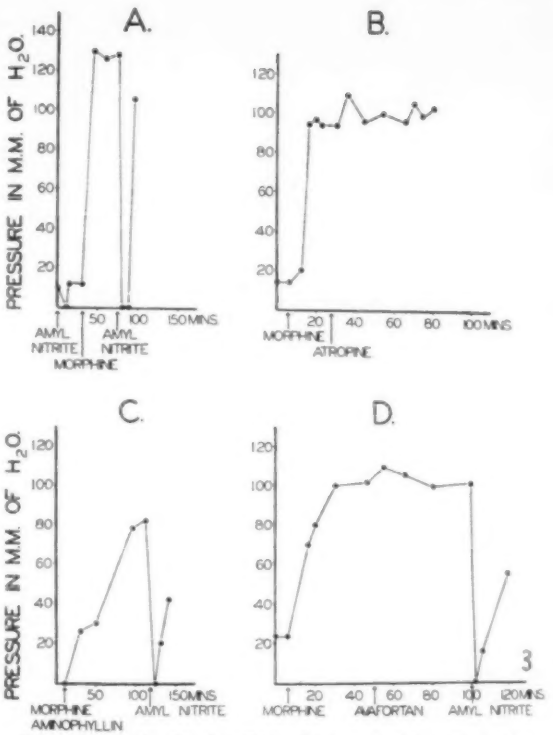

Fig. 3. Graphs showing the effects of (A) amyl nitrite, (B) atropine, (C) aminophylline and (D) avafortan on the intrabiliary pressure, following an injection of morphine.

biliary pressure rises. Amyl nitrite was given at the end of the experiments simply to ascertain that the apparatus was functioning properly. Intramuscular aminophylline has the disadvantage of being a painful injection.

Intramuscular avafortan (Fig. 3D) has no effect on the raised intrabiliary pressure produced by morphine. In some patients avafortan relaxes the tone of a normal sphincter of Oddi.[17]

None of the other antispasmodics tried in combination with morphine have any effect on the spasm of the sphincter of Oddi produced by the morphine (Table II). Therefore, at present avafortan — an analgesic that does not produce spasm

TABLE II. THE EFFECTS OF VARIOUS ANTISPASMODICS ON SPASM OF THE SPHINCTER OF ODDI PRODUCED BY MORPHINE

Drug	*No. of administrations*	*Degree of relaxation of spasm produced by morphine*			
		Nil	*Min.*	*Mod.*	*Marked*
Amyl nitrite	52	0	0	0	52
Aminophylline	5	1	1	3	0
Glycerine trinitrite	3	2	1	0	0
Atropine	6	6	0	0	0
Avafortan	6	5	1	0	0
Buscopan	3	2	1	0	0
Bentyl	4	3	0	0	1
Khellin	2	2	0	0	0
Papaverine	1	1	0	0	0
Priscol	2	2	0	0	0
Daptazole	3	3	0	0	0
Scopolamine	2	2	0	0	0
Ephedrine	2	2	0	0	0
Nalorphine	3	2	0	0	1

Min. = minimal, Mod. = moderate.

of the sphincter of Oddi — appears to be the best drug in the treatment of biliary colic due to a gallstone in the common bile duct.

The potency of avafortan is apparently equivalent to that of pethidine. Intramuscular avafortan has been tried in 3 cases of biliary colic caused by stones in the common bile duct with good results in 2 patients and an indifferent result in the third. Since the manufacturers advise that in cases of severe colic avafortan is more effective if given intravenously, it is possible that intravenous avafortan would have been more effective in our third patient.

EFFECT OF DRUGS ON THE GALLBLADDER

The effects of these drugs on the gallbladder were then investigated.

Morphine

Morphine is said to cause contraction of the gallbladder.[7,21] This conclusion was based on the following experiment:

A de Pezzer catheter or T-tube was inserted into the fundus of a gallbladder in a dog. The long limb of the T-tube was brought out through a stab wound in the abdominal wall and the abdomen closed. After the dog had recovered from the operation, a water manometer was attached to the catheter and the effect of morphine was tested with the dog at rest.

Fig 4 shows a graph of the pressure studies in this experiment. The resting pressure in this dog's gallbladder

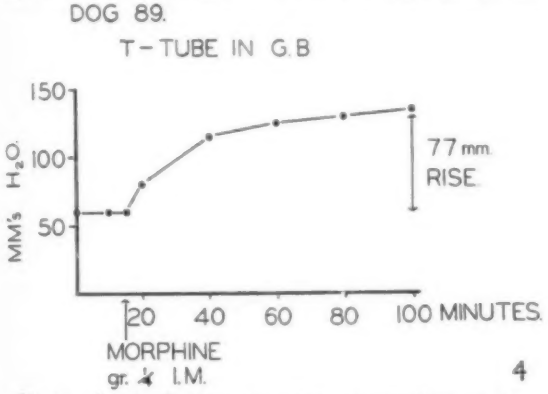

Fig. 4. Graph showing the effect of morphine on the intra-gallbladder pressure in a dog. (G.B. = gallbladder. I.M. = intramuscularly.)

was 60 mm. of water. Morphine, gr. ¼, was given intramuscularly. This resulted in a rise in intra-gallbladder pressure by 77 mm. of water. A similar rise in gallbladder pressure was obtained with pethidine.

There are 2 possible explanations for the rise in gallbladder pressure after the morphine injection (Fig. 5). Firstly, Macht[7] concluded that the rise in pressure was due to the contraction of the gallbladder itself. It is noteworthy that he did not ligate the cystic duct and did not carry the experiment further. A second possible explanation for the rise in pressure could be back pressure transmitted up the cystic duct from the raised intra-common-duct pressure produced by spasm of the sphincter of Oddi.

It was therefore felt that the experiment should be repeated after ligation of the cystic duct, but with the

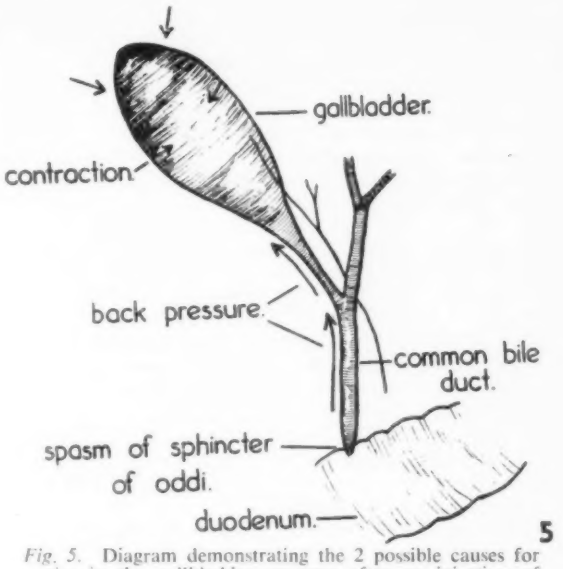

Fig. 5. Diagram demonstrating the 2 possible causes for a rise in the gallbladder pressure after an injection of morphine.

blood supply to the gallbladder intact. After ligation of the cystic duct morphine had no effect on the gallbladder pressure (Fig. 6A). In order to ascertain that the blood supply to the gallbladder was unimpaired after ligation of the cystic duct the dogs were given pancreozymin, a pancreatic hormone which contains cholecystokinin.[22] The

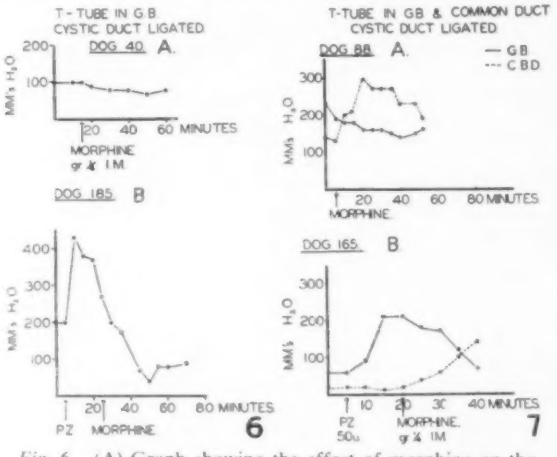

Fig. 6. (A) Graph showing the effect of morphine on the gallbladder of a dog after ligation of the cystic duct. (B) Graph showing the effects of pancreozymin and morphine on the gallbladder of a dog after ligation of the cystic duct. (G.B. = gallbladder, I.M. = intramuscularly, P.Z. = pancreozymin.)

Fig. 7. (A) Graph showing the effect of morphine on the gallbladder and the common-duct pressure following ligation of the cystic duct. (B) Graph showing the effects of pancreozymin and morphine on the gallbladder and the common-duct pressure following ligation of the cystic duct. (G.B. = gallbladder, CBD = common bile duct, PZ = pancreozymin, I.M. = intramuscularly.)

intravenous injection of pancreozymin (Fig. 6B) produced a short-lived but marked contraction of the gallbladder, lasting approximately 20 minutes.[23,24]

Here again there was no rise in gallbladder pressure after the injection of morphine. In fact, the pressure fell below the resting pressure, suggesting that the gallbladder may indeed have relaxed.

It remained to be shown that although there was no rise in pressure in the gallbladder after ligation of the cystic duct, there was indeed a simultaneous rise in common-bile-duct pressure. After ligation of the cystic duct, T-tubes were inserted into the gallbladder and into the common bile duct. Subsequently pressures were measured.

Following an injection of morphine (Fig. 7A) the pressure in the common bile duct rose to 140 mm. above that of the gallbladder pressure. The gallbladder pressure gradually fell after the morphine injection.

An attempt was then made to reproduce an attack of gallbladder colic in the experimental animal to observe the effect of morphine on the pressures in the biliary system in these circumstances.

A dog with T-tubes inserted into the gallbladder and common bile duct and whose cystic duct had been ligated, was given 50 units of pancreozymin, to produce acute gallbladder contraction. The graph in Fig. 7B shows that after the injection the pressure in the gallbladder rose steeply, whereas the common-duct pressure remained unchanged. At the height of the contraction ¼ gr. of morphine was given intramuscularly. The common-duct pressure rose steadily to above the gallbladder pressure, while the gallbladder pressure fell after the effect of the pancreozymin had worn off.

These experiments seemed to prove that morphine does not cause contraction of the gallbladder and perhaps even relaxes it, the rise in gallbladder pressure after morphine being due to back pressure up the cystic duct from the raised intra-common-duct pressure, caused by spasm of the sphincter of Oddi. It was thought possible that the back pressure in the cystic duct after the injection of morphine could disimpact a gallstone from the neck of the gallbladder and, with this in view, the following experiment was performed.

TABLE III. THE EFFECT OF MORPHINE ON THE HUMAN GALLBLADDER IN 6 CASES

Patient	*Effect of morphine on gallbladder*	*Effect of a fatty meal after morphine*	*Remarks*
1. L. B.	Increased distension	Increased distension	
2. M. J.	Slight contraction	No contraction	Patient vomited after morphine? Cause of contraction
3. S. G.	No contraction	No contraction	
4. L. P.	No contraction	No contraction	
5. J. B.	No contraction	No contraction	Gallbladder contracted following 2nd fatty meal 4 hours after morphine
6. F. de G.	Increased distension	Increased distension	

The gallbladder in a dog was mobilized from the fundus down and circumcised at the neck after the insertion of stay sutures. A close-fitting human gallstone was inserted into the neck of the gallbladder, making sure that it was not impacted and that it could be dislodged fairly easily (Fig. 8). The gallstone in the neck of the gallbladder was observed for 10 minutes under basal conditions.

Fig. 8. Diagram of experiment in a dog showing a human gallstone inserted into the neck of the gallbladder.

The dog was then given ¼ gr. of morphine. Within 3 - 10 minutes, depending on the method of administration of the morphine, the stone was dislodged from the neck of the gallbladder.

This experiment was performed several times with the same result and it therefore seems reasonable to conclude that, given the correct conditions, morphine may dislodge a gallstone from the neck of the gallbladder in a dog.

In order to assess the value of the conclusions drawn from the above experiments performed on the gallbladders of dogs, it was necessary to determine whether the human and canine gallbladders react in the same way to drugs. This was investigated in the following way:

Six patients having routine cholecystograms, whose gallbladders had been filled adequately with dye, were given ¼ - ½ gr. of morphine intramuscularly. They were re-X-rayed from ½ to 2 hours after the morphine injection to observe the effect. The patients were then given fatty meals at varying intervals to see the effect of a stimulant on gallbladder contraction. Similar X-ray views were taken throughout the series.

Table III shows the effect of morphine on the gallbladder in the 6 patients. There was no contraction of the gallbladder after the morphine injection, except in patient 2 (M.J.). She vomited after the morphine was administered and this may have caused the gallbladder to contract and empty. Following morphine, the gallbladder failed to contract after a fatty meal in all cases.

In patient 5 (J.B.), the gallbladder had not altered in size ½ an hour after the injection of morphine. Similarly the gallbladder did not contract after the fatty meal. However, 4 hours after the morphine injection the fatty meal was repeated and on this occasion the gallbladder contracted, i.e. after the effect of the morphine had worn off.

In order to exclude delay of the fatty meal reaching the duodenum due to gastric stasis produced by morphine, a tube was passed into the duodenum in 3 cases and the fatty meal injected directly into the duodenum. Even under these circumstances the gallbladder did not contract.

From this it was concluded that morphine did not make the human gallbladder contract and appeared to inhibit the contraction normally produced by a fatty meal.

Atropine

It has been said that atropine causes the gallbladder to relax.[25] The effect of atropine on the human gallbladder

was tried on 4 patients who were having routine cholecystograms. Each patient was given $\frac{1}{100}$ gr. of atropine intramuscularly and re-X-rayed 15 minutes after the injection. This was followed by a fatty meal, and a further X-ray. In 3 cases the gallbladder decreased in size after atropine and in 1 case the gallbladder increased in size. In all 4 patients the gallbladder contracted in response to the fatty meal.

From these results it seems that atropine does not inhibit contraction of the gallbladder produced by a fatty meal and therefore is probably not of value as an antispasmodic in gallbladder pain.[29]

CONCLUSIONS

From these observations the following conclusions in regard to the treatment of biliary pain seem justifiable:

1. If the pain is due to a common duct stone, avafortan is the drug of choice. It is analgesic and does not produce spasm of the sphincter of Oddi. However, a more potent analgesic and antispasmodic drug would be preferable.

2. If the pain is due to a stone impacted in the neck of the gallbladder, morphine is the drug to be used. It is a powerful analgesic and does not cause contraction of the gallbladder. Indeed, it may even inhibit gallbladder contraction. Furthermore, due to contraction of the sphincter of Oddi, a rise in common-bile-duct pressure is caused which may be transmitted *via* the cystic duct and may result in disimpaction of the stone with complete remission of symptoms.

SUMMARY

1. The effects of various analgesics on the sphincter of Oddi, alone and in combination with various antispasmodics, are shown.

2. Of all the analgesics tested only avafortan does not produce spasm of the sphincter of Oddi.

3. The effect of morphine on the gallbladder is demonstrated.

4. Morphine does not cause the gallbladder to contract and perhaps even inhibits contraction. Furthermore, it was shown in dogs that spasm of the sphincter of Oddi produced by the morphine results in a rise in the common-duct pressure which may be transmitted up the cystic duct and dislodge a gallstone from the neck of the gallbladder.

5. It is suggested that in the treatment of biliary pain, if the pain is due to a common duct stone, avafortan is the drug of choice since it is an analgesic that does not produce spasm of the sphincter of Oddi. However, if the pain is due to a stone impacted in the neck of the gallbladder, morphine should be used.

A powerful analgesic which does not cause contraction of the gallbladder, morphine raises the common duct pressure, and this rise in pressure may be transmitted up the cystic duct to result in disimpaction of the stone with relief of symptoms.

I wish to thank Dr. J. G. Burger, Medical Superintendent, Groote Schuur Hospital, for permission to publish details of the cases mentioned, and Prof. J. H. Louw of the Department of Surgery, University of Cape Town, for his interest and advice on the preparation of this paper. Thanks are also due to the Director of Surgical Research, Dr. C. N. Barnard, for making laboratory facilities available, and to Dr. L. Werbeloff and his staff for their kind help with the radiological aspect of this work. I am grateful to Mrs. E. P. Kottler and Mr. G. McManus for the diagrams and photographic work.

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POSTOPERATIVE INTESTINAL OBSTRUCTION IN INFANTS AND CHILDREN*

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An analysis of experience with intestinal obstruction following abdominal operations in infants and children has provided some unexpected results in regard to the causes of the obstruction; and, although the condition is not common and the experience reported here is limited, it appears to be worthy of record.

* Paper presented at the Second Congress of the Association of Surgeons of South Africa (M.A.S.A.), Durban, 17 - 20 September 1960.

The material studied consists of 20 cases which have arisen in the course of the past 9 years. The 20 cases all presented a mechanical intestinal obstruction; they had all had previous abdominal surgery; and they were all submitted to further surgical treatment — thus a definite diagnosis could be made. These were not the only instances of postoperative obstruction that occurred during this period; there were also examples of paralytic ileus as well as cases of mechanical ileus which were treated conserva-

tively; these cases are listed in Table I to provide a perspective of their relative incidence. The absolute incidence of the conditions cannot be given with any degree of statistical accuracy, since the material was derived from a number of different sources and the treatment was carried out in several different hospitals.

TABLE I. RELATIVE INCIDENCE OF POSTOPERATIVE INTESTINAL OBSTRUCTION

Obstruction	*How treated*	*No. of cases*
Paralytic ileus:		
with peritonitis	—	14
non-infective	—	4
Mechanical ileus	Conservatively	12
Mechanical ileus	By operation	20

It is highly probable that the 12 cases of mechanical ileus, which were treated conservatively, were caused by postoperative adhesions, but they are omitted from this study because the diagnosis was not made with precision and certainty. This paper is confined to a report on the 20 cases of mechanical ileus treated by operation and is concerned with the proved diagnosis of the causes of the postoperative mechanical obstruction.

The age groups into which these cases fall (i.e. at the time of their postoperative intestinal obstruction) are as follows: neonatal, 4 cases; under 2 years, 4 cases; and over 2 years, 12 cases.

NEONATAL

Case 1

The original operation was for malrotation of the gut performed on the second day after birth; postoperatively the obstruction persisted, and re-exploration on the sixth day revealed the presence of a band constricting the duodenum.

Case 2

The original operation was described as having been done for a volvulus of a midgut loop on the third day of life; evidence of obstruction recurred on the 14th day, and at re-operation 3 days later, a recurrent volvulus of the whole midgut loop was discovered.

Case 3

The initial operation was for a congenital abnormality of the common bile duct; 3 weeks later, multiple adhesions were freed at the second laparotomy.

Case 4

The original condition was a tracheo-oesophageal fistula with a blind, short oesophagus. Repair and reconstitution was effected *via* a combined thoracotomy and laparotomy; postoperatively, intestinal obstruction became manifest, and on the 5th day a further laparotomy showed malrotation of the gut with obstruction of the third part of the duodenum.

It is noteworthy that the postoperative obstruction was due to adhesions in only 1 case out of the 4 in this group.

UNDER 2 YEARS

Case 1

At the first operation, done at the age of 14 months, it was reported that an acutely inflamed appendix was removed. A number of episodes of abdominal pain followed over the succeeding months, and at the age of 19 months an acute complete obstruction presented; on the 3rd day of this obstruction, an 8-inch-long segment of reduplication of small bowel was resected.

Case 2

This was of a similar nature. About 2½ months after appendicectomy, re-exploration for intestinal obstruction revealed a mesenteric cyst at a fairly high level of the jejunum.

Case 3

This infant of 5 months of age was referred 4 days after an operation at which an ileo-caecal intussusception was reduced. Persistent obstruction called for a second operation, at which a second intussusception, some 4 feet proximal to the ileo-caecal junction and caused by a Meckel's diverticulum, was found.

Case 4

A persistent vitelline duct, which had discharged at the umbilicus since birth, was dissected out and removed at the age of 5 months; at this operation, the ileal end of the fistula was carefully examined, but no other abnormality was noted at the time.

Some 5 weeks later, after a clinical picture of intestinal obstruction lasting for about 12 hours, re-operation showed distension of the small bowel proximal to the level of the previous site of closure of the ileum; distal to this site, the ileum was collapsed. The level at which distended bowel met collapsed bowel was abrupt, but there was no evidence of an organic obstruction at the site. A side-to-side anastomosis was done, short-circuiting the affected loop; this led to relief of the condition.

OVER 2 YEARS

Case 1

This case was very similar to case 4 of the 'under-2-years' group. A girl, aged 2 years and 8 months, was reported to have had a resection of a Meckel's diverticulum 21 days before the onset of a bout of intestinal obstruction; this obstruction appeared to relent, and subsequent X-ray studies showed a partial hold-up in the lower ileum; 2 further episodes of obstruction occurred and then, 6 weeks after the first operation, another, and apparently complete, obstruction supervened.

At the second operation, a condition similar to that described in case 4 in the 'under-2-years' group was found; there was marked distension, of a spherical type, at the site of the removal of the diverticulum; the distended portion of bowel was about the size and shape of a tennis-ball, sharply limited at its distal end and fading somewhat into moderately distended bowel proximally. About 8 inches of bowel, including the distended portion, was removed and continuity was re-established by end-to-end anastomosis. The resected portion of bowel did not show any mechanical obstructing factor, nor was the histological examination suggestive of any form of nerve-plexus abnormality.

Cases 2 and 3

There were 2 patients, one aged 7 and another aged 9 years, in whom an appendicectomy had been done, respectively, 2 weeks and 5 weeks previously. The obstructions in these 2 cases were both due to intraluminal plugs. In one, an undigested bolus of fruit, and in the other, a firmly-packed plug of round worms, was found.

Cases 4 - 8

Five patients, with previous appendicectomies, presented with subsequent intestinal obstruction which was found to be due to:

(*a*) an intussusception in the lower ileum;
(*b*) a jejuno-jejunal intussusception due to an adenoma;
(*c*) an adherent Meckel's diverticulum causing acute kinking of the ileum;
(*d*) a volvulus of the small bowel caused by a Meckel's diverticulum; and
(*e*) a large mesenteric cyst causing compression of the ileum.

Apart from case (*e*), the mesenteric cyst, the other 4 postoperative obstructions presented within 6 weeks of the initial operation; that due to the mesenteric cyst occurred 11 weeks after the appendicectomy.

Case 9

A boy of 7 years had a repair of an hiatus hernia 8 days before the onset of an intestinal obstruction. The original operation had involved a combined thoraco-abdominal explora-

tion. Re-operation showed the cause of the obstruction to be malrotation of the gut, with loops of bowel lying under the right cupola of the diaphragm, above the right lobe of the liver.

Cases 10 - 12

In 2 of these, a previous appendicectomy had been done, respectively, 6 months and 3 years before the obstruction. In the third case, a Meckel's diverticulum had been operated upon 18 months before the obstruction. They all proved to be due to adhesions affecting mainly the lower loops of the ileum.

DISCUSSION

In 3 cases, the last 3 listed above, the obstruction may be said to have appeared late in the postoperative period. They were cases where firm, well-established adhesions caused the obstruction. All the other cases, in all 3 age groups, arose at a relatively early postoperative stage. Only 1, in the neonatal group, was caused by adhesions; in the remainder, the findings were rather unexpected in that they were due to pathological conditions other than adhesions. In 3, possibly 4, cases the condition was either a part of, or a recurrence of, the original condition for which the first operation had been done, but in no fewer than 12 cases the obstructing factor was unrelated to the original condition and suggested either a second pathology or, in fact, a primary pathology which was mis-diagnosed and overlooked at the time of the initial laparotomy.

It is usually held that early postoperative intestinal obstruction in infants and children is most commonly due to early adhesions, of a soft and plastic variety, not requiring surgical intervention, but calling for conservative management. This series of cases, admittedly small, suggests that a modified attitude is required; a significant number of such early postoperative obstructions is due to distinct and surgically amenable lesions. It may be argued that the initial surgery was inadequate or careless in many, or most, of the cases; however, it is to be noted that, for the most part, the initial treatment was performed or supervised by experienced and competent surgeons.

SUMMARY

1. A series of 20 cases of intestinal obstruction following abdominal operations in infants and young children is presented.

2. In 16 of these cases the obstruction was due to conditions other than postoperative adhesions; 17 of the cases arose in the early postoperative period.

3. In 12 of the cases, the condition causing the obstruction was quite unrelated to that for which the original abdominal operation was performed.

4. It is suggested that a modified attitude to postoperative intestinal obstruction is needed, since in a significant number of cases the early postoperative obstruction is due to a distinct and surgically treatable condition.

ASSOCIATION OF SURGEONS OF SOUTH AFRICA (M.A.S.A.)

SUMMARIES OF SCIENTIFIC PAPERS*

1. SOME OBSERVATIONS ON PAGET'S DISEASE OF THE NIPPLE

MR. C. TOKER, *University of the Witwatersrand*

Paget's disease of the nipple is the association of a carcinoma in the portion of one or more lactiferous ducts within the nipple and an eczema of the skin of the nipple. The eczematous nipple skin contains within it large cells—the Paget cells. In the opinion of many authorities the Paget cells represent duct cancer cells that have directly invaded the epidermis. The responsible tumour is situated within the nipple ducts close to their orifices, and not deeply within the breast substance.

The study reported here was based upon the detailed examination of a single case of Paget's disease. The carcinoma that had produced the Paget infiltrate was not located superficially within the ducts but within the breast substance, 3 cm. below the surface. The cancer cells had spread up to the epidermis within the mucosal lining of a single duct system. This fact was established by serial section of the entire nipple. The tumour cells occupied the plane of the basal layer of the mucosa, and had left undisturbed both the superficial layer of the duct lining and the basement membrane. This mode of spread has been called intra-epithelial permeation.

No previous record had been found of the production of a clinical Paget lesion by ascent of tumour cells from a focus situated deep within the breast substance, nor of spread of this nature over so considerable a distance.

2. ASPECTS OF HEAD INJURIES—EXPERIMENTAL AND CLINICAL

MR. D. A. MUSKAT, *Johannesburg*

The immediate problems presented in grave cerebral injuries are: (1) Is there an immediately remediable lesion? (2) If not, what are the best methods of promoting vegetative survival and possible recovery?

The first problem depends for its answer on whether the lesion is operatively or physiologically removable. Should it be operatively removable, how can it be decided diagnostically whether operative intervention is indicated? The so-called 'vital' signs, which are often absent in the presence of space-occupying lesions or present in their absence, are frequently misleading.

Kocher described the 'classical' changes of increasing cerebral compression, and Cushing tried to show experimentally that the 'vital' signs did occur. He required cerebrospinal-fluid pressures equal to the blood pressure to elicit the 'vital' responses. However, as is well known, such high pressures rarely, if ever, occur clinically. Browders and Meyers, in clinical and experimental observations, were also very sceptical about the value of the 'signs' of compression.

No record could be found of earlier experiments to simulate pathologically-occurring space-occupying lesions. A series of animal experiments was reported in which a measured quantity of blood was introduced into the various intracranial 'compartments', viz. extradural, subdural, combined, subarachnoid, intracerebral and intraventricular. The effects of these 'haematomata' on the blood pressure, cerebrospinal-fluid pressure, pulse-rate and respiration were recorded up to the time of the death of the animal. (Dogs were used in all these experiments.) The responses elicited varied so much from animal to animal that the significance of any one finding (e.g. rise in blood pressure) could not be correlated with that of another 'vital' change. These were claimed to be the first animal experiments designed to prove that the Kocher-Cushing dicta, so long accepted, do not apply, at least as far as rapidly increasing intracranial compression is concerned.

That reflex vasovagal effects are produced was not denied. Disturbances consequent to severe cranio-cerebral trauma evolve in 2 phases, viz. (1) vagotonic (pallor, low blood pressure, bradycardia, hypothermia) possibly due to the

* Papers presented at the Second Congress of the Association of Surgeons of South Africa (M.A.S.A.), Durban, 17-20 September 1960.

liberation of acetylcholine in the CSF and (2) sympathicotonic (tachycardia, tachypnoea, increased metabolism and febrile disturbance). These phases could well superimpose themselves on purely compressional effects. The clinical and prognostic value of the 'classical' signs are too often assiduously misapplied. There therefore remains only one clinical means of evaluation and that is the state of consciousness, which must be closely watched, ideally by the physician at the bedside, next best by a well-trained or instructed nurse. A specially designed 'chart of the conscious state' is utilized.

A comprehensive classification of the haemorrhages in and around the brain was shown and a resumé given of the modern methods of treatment of severe head injuries, with special emphasis on electrolyte disturbances and their control. Included were tracheotomy, hypothermia, steroid and vitamin therapy, and the judicious employment of a new dehydrating agent, 'urevert'.

A series of slides was shown to illustrate the 'intracranial haematomata' and their physio-pathological results, side by side with actual pathological cases with corresponding lack of 'vital' signs.

3. A NEW TECHNIQUE IN THE DIAGNOSIS OF HIRSCHSPRUNG'S DISEASE

MR. B. SHANDLING, *University of Cape Town*

The modern histopathological concept of the underlying abnormality of innervation of the gut was discussed, and the clinical presentation of Hirschsprung's disease was arbitrarily classified into 2 broad groups—(1) in the neonatal period, and (2) in the older child.

It is in the neonate presenting with signs and symptoms of intestinal obstruction that diagnostic difficulty is encountered, and in such cases opaque enema studies are mandatory. Unfortunately, these are not always completely unequivocal, since mistakes may arise in the 'short-segment' type of Hirschsprung's disease. Also, in the newborn baby the disproportion between the normally ganglionated and aganglionic segments may not have developed to any marked extent.

A biopsy of the rectum to include the muscle layers will, by the absence or presence of ganglion cells, prove or refute the diagnosis of Hirschsprung's disease.

The previously reported methods of performing a rectal biopsy were discussed—those of Swenson, Bodian, and Hiatt. They all require a general anaesthetic, necessitate incisions and suturing of layers, and are subject to the complications of haemorrhage and sepsis.

A study of the anatomy of the transverse rectal folds or valves of Houston revealed the presence of both muscle layers of the rectum in their structure. Using an insufflatory biopsy technique, with a specially modified sigmoidoscope eyepiece, and cutting multiple sections of the material so obtained, biopsies in 32 normal children showed the presence of ganglion cells in every case. Eight patients with Hirschsprung's disease showed complete absence of ganglion cells.

This method involves no general anaesthetic; no incisions requiring suturing are necessary; it cannot possibly interfere with a subsequent Swenson or Duhamel operation; and it has in no case been complicated in any way by haemorrhage, sepsis, or fibrosis.

With an adequate biopsy of the valves of Houston the diagnosis of Hirschsprung's disease can be confirmed or refuted.

4. SOME OBSERVATIONS ON THE AETIOLOGY OF GALLSTONES

MR. T. G. LORENTZ, *University of the Witwatersrand*

A general review of the incidence of gallstones was given, emphasizing the difference in sex and racial incidence.

The possible theories of causation were then reviewed under the headings of (1) stasis, (2) infection, and (3) an altered biochemistry of the bile.

The physiology of cholesterol and its relation to the bile acids was briefly discussed and it was suggested that the altered bile-salt/cholesterol ratio necessary for stone formation might have its origin in the liver by a depression of bile-acid synthesis.

An investigation of gallbladder pathology in the monkey was described, in which it was found that 10% of the gallbladders examined showed evidence of cholecystitis. No calculi were observed, presumably due to a favourable bile-salt/cholesterol ratio.

A preliminary account was then given of an investigation into the dietary production of stone in hamsters. This followed the work of Dam in Sweden and demonstrated the production of cholesterol stones on a low fat and cholesterol diet. Here the possible mechanism was discussed and thought to be biochemical in origin. Failure to produce stones in the South African hamster—mystromus—was observed.

5. OBSERVATIONS IN OESOPHAGEAL SURGERY

MR. D. S. CHAPMAN, *University of Natal*

A trial of various forms of colon by-pass for oesophageal cancer, using the retrosternal tunnel, was reported and contrasted with the same technique employed for extensive, impassable strictures.

Excluding adenocarcinoma (rare in any series in the African), the commonest site of oesophageal cancer is the most inaccessible and dangerous upper thoracic half of the oesophagus. Not even using very rigid criteria, only 50% of all cases are operable and only 20 - 30% are resected. In only 15 - 20%, where lymph nodes and local structures are not apparently involved, is the operation deemed 'curative'. Few series have acceptable operative mortality rates and a recent world review has estimated an average operative mortality of 30% for all sites. Lastly, despite an occasional claim of cure, no matter what is done the survival rate is appalling.

It was on this evidence that it was decided to perform, as a first stage, a by-pass well away from the cancer to permit of a more rapid improvement of nutrition than had previously been obtained by the usual methods; then soon afterwards to attempt curative resection through a thoracotomy. It was hoped that, should such a resection be abandoned on grounds of spread, palliation would be assured.

With due regard to the usual site of occurrence, the proved extensive mural spread far beyond the usually accepted range, and the requirements of oesophageal blood supply, it was strongly advised for any hope of cure that the right thoracic approach be used in all cases other than cancer of the cardia, and that removal of the whole oesophagus and anastomosis in the neck be done, whether the patient was prepared by means of a by-pass or not.

The detail necessary in gastric mobilization and preparation for anastomosis, where the stomach was preferred for restoration, was commented on. Some facets of the operative technique using the colon by-pass were outlined, with special reference to anatomical vascular and anastomotic details.

In common with other recent papers, this report suggested that the trial of the use of colon by-pass in the treatment of oesophageal cancer was a failure and condemned the use of such a method for palliation alone. Although there were only 2 immediate deaths in 15 cases, 10 of the 13 survivors developed either cervical or abdominal fistulae or both.

This exceptionally high and disabling morbidity was contrasted with the excellent uncomplicated palliation obtained by the same technique in 5 cases of extensive impassable stricture.

Should ablation be preferred for cancer, a most careful selection is required at thoracotomy and unless, at least, the oesophagus is able to be mobilized with ease, the case certainly has incurable spread. A resection is thus rarely merited. A by-pass operation, so excellent a palliation for the benign stricture, is to be condemned as a palliation for cancer when simple methods of intubation are available. In many cases the attempt to use an otherwise attractive technique for the preparation of cases for resection had proved a failure. Radiotherapy gives only slightly better results for cancer of the upper oesophagus and is inferior to surgery for growths near the diaphragm.

6. SURGICAL ASPECTS OF BILHARZIASIS

PROF. A. E. KARK, *University of Natal*

Schistosomiasis is endemic in the coastal region of Natal and the surgical aspects of the disease constitute a serious clinical problem. The disease has unfortunately been regarded as benign, but in Indians and Africans the complications of stricture and carcinoma occurring at an early adult age are serious, leading to progressive renal failure.

In the overwhelming majority of cases the lower urinary tract is involved and the parasite is usually *Schistosoma haematobium*. The mere presence of ova, whether dead or alive, signifies that the disease is present. The essential change is one of subepithelial hyperplasia which may heal or progress to polyp formation, calcification and infection.

1. *Bilharzial Cystitis*

The commonest manifestation is bilharzial cystitis where haematuria is the outstanding symptom. Ova are found in less than 20% of cases unless examination is carried out repeatedly. The only certain method of diagnosis is by cystoscopy. Calcification is seen in 10% of cases, in many of which there is little contraction of bladder capacity despite extensive calcification.

The characteristic cystoscopic picture is a proliferative and non-ulcerative tubercle-like lesion with a background of ground-glass pallor in the later stages. With secondary infection there is a wide-spread cystitis and polyp formation.

2. *Drug Therapy*

The most satisfactory drug has proved to be 'triostam', 225 mg. given daily by intravenous injection for 6 days; repeat cystoscopy in 3 weeks in the majority of cases shows complete response.

3. *Right-iliac-fossa Syndrome*

A particular syndrome of pain in the right iliac fossa, with or without a history of urinary difficulty, frequency or haematuria, is a frequent clinical presentation closely resembling acute appendicitis. It is commoner on the right side and in young men in their early twenties, more so in Indians than Africans. The general signs of acute appendicitis are absent. Full urinary investigation is necessary and, if negative, should be repeated in 3 months to determine the presence of early bilharzial ureteritis.

4. *Bilharzial Ureteritis and Strictures*

Eighteen strictures were treated in Indians and only 1 in an African male. The majority were at the lower end of the ureter and in a third of them urinary concretions had accumulated behind the stricture. The effects in the ureter result from fibrosis of the orifice with proximal dilatation or from wide-spread bladder disease with reduction in capacity and secondary ureteric dilatation. The latter is rare in Durban. The symptoms of stricture resemble renal colic. Full investigation will reveal either a persistent filling of the lower segment or there may be extensive dilatation with irregular beading. When a catheter cannot be passed a uretero-pyelogram should be done with the ureteric opening blocked.

If possible ureteric dilatation should be attempted and failure to pass a No. 8 catheter is suspicious of stricture. Cystoscopic meatotomy has been advised but has not been used in Durban with advanced strictures. Excision and re-implantation of the ureter should be undertaken following a full course of triostam. The 'roll-back' technique with mucosa-to-mucosa suturing has proved satisfactory for strictures within an inch of the bladder. By this means an attempt, often successful, is made to provide a valve and so prevent reflux.

5. *Hydronephrosis*

The incidence here is low, hydronephrosis being present in approximately 20% of the cases with cystitis, but in a third of the cases with carcinoma. Where possible the primary cause of the hydronephrosis should be treated. The fact that 12% of Africans showed hydronephrosis, yet only 1 stricture was seen, suggests that intravesical pressure increase and widespread destruction of the ureteric walls are at least as important as stenosis as a cause of renal dilatation.

6. *Carcinoma*

In 5 years 52 cases were seen, 36 in Africans and 16 in Indians, the sex ratio being approximately 3 : 1. Bilharzial ova were present in 60%. Histologically the striking feature is the high incidence of squamous lesions (50%); the majority had ova and showed evidence of keratinization. Bilharzial papillomata are not regarded as premalignant, but leukoplakia, which has seldom been noted in this series, is so regarded.

The argument against the rôle of bilharzia in producing bladder carcinoma is based on the rarity of bowel carcinoma, despite heavy rectal bilharzial infestation, and the equal rarity of ureteric or lung carcinoma, despite deposition of ova. Yet the frequency of squamous lesions and metaplasia is strong evidence of causal association, and it has been suggested that super-added urinary infection is one of the factors in inducing neoplastic change.

The bladder neoplasms occur at a young age, 30 - 50 years. The majority are surgically incurable on admission, but urinary deviation is a valuable palliation.

7. *Calculus*

There is a great difference in the incidence of urinary calculi in Indians and Africans. Despite the high incidence of obstructive lesions in the urinary tract, frequently due to bilharzia, the African rarely develops calculi, whereas this condition is among the commonest urinary complaints in Indians.

7. OESOPHAGEAL HIATUS HERNIA

MR. D. FULLER, *Johannesburg*

Altogether, 325 cases of sliding hiatus hernia had been seen in consultation. In many, only the usual 'medical' treatment was advised. To be effective, this must be carried out vigorously, and where necessary there must be drastic weight reduction and tight corsets must be discarded. There are many antacid substances which give good relief. Where there is oesophagitis the head of the bed must be raised to diminish free acid flow up the oesophagus during sleep. A number of patients had minor symptoms not justifying operation, while many others were not fit for operation for various reasons.

In assessing those cases needing operation, oesophagoscopy was carried out at first. This practice has now been discarded as a routine. A 'diagnostic oesophagoscopy' was performed in 31 cases. A further 38 patients had oesophagoscopy for the dilatation of a stricture resulting from old-standing oesophagitis. A number of these needed frequent dilatations.

Curative repair was done in 128 cases, while 15 needed oesophago-gastrectomy or some other operation for removal of a strictured oesophagus or, as in the case of 4 patients, for the removal of a carcinoma within the supra-diaphragmatic pouch.

The follow-up of the 128 cases has thus far revealed 7 recurrences (5·5%). Of these, 3 have undergone a second operation with a second recurrence in 1 case. In patients operated upon by other surgeons, 21 recurrences have been seen, and 9 of these have had a further operation with a second recurrence in 1 case.

Of the two common approaches to the oesophageal hiatus the trans-thoracic, supra-diaphragmatic one was preferred. This has been used in all except 2 cases. Anatomical visualization of the region is generally better from above and the planned operation can be satisfactorily completed. In the many cases of dense peri-oesophagitis and the few associated with other oesophageal lesions, the trans-thoracic approach is the only one that can provide adequate access. There is no doubt that in the usual reducible sliding hernia, repair from the abdomen can be just as satisfactory if adequately performed. In most of the 21 recurrences, where the first operation was performed elsewhere, the symptoms remained unchanged after operation, indicating inadequate reduction and repair initially. Of the 17 recurrences in the author's series, all but 1 were free of symptoms for months or years after operation.

Apart from the usual association of cholelithiasis and duodenal ulcer in many of these patients, the following interesting lesions were encountered: carcinoma in the gastric pouch, 4 cases; carcinoma of the lower oesophagus, 1 case; leiomyoma of the cardio-oesophageal junction, 3 cases;

leiomyoma of the fundus of the stomach, 1 case; submucous cyst of the oesophagus, 2 cases; and Morgagni hernia, 1 case.

Fifteen cases had resection for stricture or associated carcinoma. The types of operation performed were: Belsey's gastric tube (oesophago-gastrectomy) with pyloroplasty, 6 cases; Belsey's gastric tube (oesophago-gastrectomy) with gastro-enterostomy, 1 case; oesophago-jejunostomy, 5 cases; and oesophago-gastrectomy for carcinoma, 3 cases. Two of these patients died as the result of the operation.

For the usual repair, a modification of Allison's technique had been used. However, the operation does not appear to be wholly satisfactory and for some months the author had combined Allison's repair with a modification of the Boerema concept using the phreno-oesophageal ligament to anchor the oesophagus an inch or two below the hiatus onto the under surface of the diaphragm. At the same time the oesophagus is tacked to the crura, which themselves are closely approximated around the oesophagus. In some patients the oesophagus appears truly short and requires to be mobilized to below the aortic arch before the cardio-oesophageal junction can be brought into the abdomen. The recurrence rate among these cases is inevitably higher and an associated gastric emptying procedure such as pyloroplasty or gastro-enterostomy with a vagotomy may be wise. In others resection is necessary. A pedicled intestinal graft has not been used to lengthen a short oesophagus or replace a strictured one in these cases.

THE TREATMENT OF MENTALLY ILL PATIENTS

By Our Parliamentary Correspondent

The Minister of Health, Dr. Albert Hertzog, told Parliament, when he replied to the recent debate on the Mental Disorders Amendment Bill, that there was no longer overcrowding at mental institutions for European patients and that steps were being taken to overcome the overcrowding at mental institutions for non-Europeans.

Dr. Hertzog said that, as far as White patients were concerned, the position had improved so much that there was room for everybody. 'In the case of non-Whites, however, we do still have overcrowding.'

'We are trying our utmost to improve the position', Dr. Hertzog continued. 'A new big mental hospital is being erected at Stikland at the moment at a cost of R5,000,000, and we hope that it will be completed next year. That alone will bring relief.

'Last year we also investigated the possibility of erecting one or more mental institutions for chronic patients in the Native areas and of then moving the chronic cases to these areas. I am expecting the report shortly when further steps will be considered.' The Bill itself, through the provision of outpatient facilities, would materially affect the position.

Shortage of Staff

The Minister admitted that 'there is a tremendus shortage of staff (to deal with mental illnesses) in South Africa, particularly of psychiatrists and specialists.'

He said: 'Our difficulties are aggravated by the fact that our training facilities are so limited, and because of that it means that we cannot go faster than the speed at which we can train specialists for our mental hospitals'. He reminded the House of Assembly that at the end of 1959 South Africa had only a few more than 70 psychiatrists.

The latest step in trying to meet this problem had been to secure the consent of the Public Service Commission for the creation of 10 clinical assistants' posts — 3 at Sterkfontein Hospital, 4 at Valkenberg Hospital and 3 at Weskoppies Hospital.

The holders of these posts would be trained as psychiatrists on condition that for every year's training they would have to serve the State for a further year. Dr. Hertzog said he hoped that in this way the State would be able to rely on each of these trainees for six years of assistance. He also expressed the hope that: 'Once they are in the service of the State they will find it so attractive that we will be able to retain the services of most of them'.

Provisions of the Mental Disorders Amendment Bill

Introducing the Bill, the Minister explained that its main provisions aimed at the establishment of outpatient facilities at mental hospitals and the extension of free treatment to voluntary as well as certified patients.

The establishment of outpatient departments at large mental hospitals would have three principal results: Firstly, large numbers of cases could be treated in the incipient stage and it was hoped that most of them would be cured. The cost to the patient, the indirect cost to his family, and the cost to the State would be small. Fewer cases would become chronic and have to be segregated for years in mental hospitals. They would in future be able to stay with their families who would care for them.

Secondly, it would be possible to discharge many patients admitted to hospitals sooner. The position now was that they had to be detained in the hospitals until practically cured. In future they could be discharged sooner and would be able to return to the outpatient department from time to time for treatment and medicines.

Thirdly, as a result of this, the congestion in the mental hospitals would be relieved and there would be more space available for other patients who would, therefore, be able to receive treatment sooner.

All these benefits would be achieved at little extra cost to the State, because no new institutions were being established — departments would simply be established at existing hospitals. The same doctors, nurses and equipment would be employed.

The extension of free treatment to voluntary patients would also help to ensure early diagnosis and treatment and prevent many cases from becoming chronic. Up to 1957 the costs of treating all mental patients could be recovered by the State from the patient's estate or relatives. In that year the principle of free treatment was accepted, but voluntary patients were excluded.

The result was that many people who felt they were in the early stages of mental aberration, delayed going for treatment until they were chronic and certifiable because they could not — or did not want to — incur the costs of treatment. By adopting a more humane attitude in the beginning, the State would have saved itself expense by being able to treat these patients timeously and being spared the costs of having to care for chronic patients.

All the doctors in the House welcomed the measure as a forward step in the treatment of mental illness, but at the same time warned that it was only a first step and that it would leave many grave problems unsolved. The chief of these was the problem of trained staff to deal with the mentally afflicted.

They all pleaded for the establishment of outpatient facilities for dealing with mental patients at general hospitals as well as at mental institutions and, in addition, some of them pleaded for the provision of beds for mental patients at general hospitals.

Discussion

Col. O. L. Shearer, M.P. for Pietermaritzburg-City, said: 'The new approach in this Bill is a very important advance. But I think we can go further'. He referred to the 'Amsterdam experiment' to demonstrate the modern approach to mental ill-health. This experiment was so successful, he said, that it cut admissions to mental institutions by 40 per cent.

The modern approach adopted in the experiment entailed taking into consideration predisposing causes, such as family environment; conditions, tensions and frustrations in the factory; and socio-economic factors. The Amsterdam city

authorities had at their disposal a specialized department to deal with the wide ramifications in predisposing causes through teamwork and coordination.

In the frontline of this joint attack was the general practitioner, the family doctor, who was in the best position to detect the slightest deviation from mental normality.

The practitioners should be specially trained through postgraduate and undergraduate courses, and practical training should be given using psychiatric beds in general hospitals where the practitioners would have contact with the patients. This training would enable the general practitioner to observe and appreciate the therapeutic approach in combination with psychologists, psychiatrists, social workers and occupational therapists.

Such an approach would ease re-adjustment in the incipient phase of mental aberration, relieve the cost to the State and the congestion in hospitals, and provide a basis for the rehabilitation of the patient in the family unit or the work atmosphere.

Colonel Shearer based his plea for outpatient facilities for mental patients at general hospitals on the stigma which he felt was attached to going to mental institutions. He quoted the report of a 1937 departmental committee on mental hospitals, which recommended the establishment of facilities at each large general hospital in the Union for the treatment of acute mental cases, to show that the idea was not a new one.

This report said: 'The principal stumbling block seems to be the financial relations between the Union Government and the provincial administrations. This difficulty, in the opinion of the committee, is not insurmountable and the committee considers the case for the establishment of such accommodation so strong that it recommends that steps should be taken towards this end at the earliest possible moment'.

Colonel Shearer urged the Minister to take up these matters with the Central Health Services and Hospitals Coordinating Committee at the first opportunity.

Dr. W. L. D. M. Venter, M.P. for Kimberley-South, was of the opinion that 'this Bill is meeting a very deep-felt need'. He added at the end of his speech: 'We can say that we are only sorry that the steps which have now been announced were not taken a long time ago .... it is essential that it should be made easier for people to receive treatment because, if we do not do so, we shall find what we find today, namely, that people are turning on a large scale to quacks in this sphere who advertise in newspapers and magazines that they can heal such people by means of hypnosis and such methods.

'Then these people fall into the hands of unscrupulous individuals who can cause them incalculable harm.

'We only hope that the progress we are now making will represent a great step towards what we should eventually like to see, namely, that outpatients of this type will be treated by every provincial hospital and not only by the mental institutions, and that far more clinic facilities will be made available so that these unfortunate people can be helped, cured and kept cured'.

Dr. E. L. Fisher, M.P. for Rosettenville, said: 'This is the beginning of a new outlook, in South Africa at any rate, in the treatment of the mentally ill'. It was the sort of thing he had been pleading for for years. He added, 'it is not going to be as easy as has been made out to treat the mentally ill in outpatients departments'.

The Minister had to make up his mind that 'If he is going to go forward with this type of legislation, he must find ways and means of providing outpatient departments in all provincial hospitals. What good would it do the people of Johannesburg or the people on the platteland to have an outpatient department attached to the Pretoria Mental Hospital at Weskoppies or to Sterkfontein?

Where would the Minister find the medical personnel to staff the clinics for outpatients? He would not be able to fill these posts by using the people at present working in mental institutions.

'I am going to tell him that, if he tries to do that, the people in the mental institutions are going to be neglected and all the medical officers and nurses are going to be overworked. Can you imagine what it means to a doctor to attend to 30 or 40 people suffering from troubles of the mind in one afternoon or in one day?'

Using general hospitals for mental diseases as well as ordinary diseases would obviate the necessity which often arose, especially in the platteland, of detaining a mentally deranged person in a police cell.

'Do not let this wonderful stride forward be obstructed by some idea that it is wrong to use an established hospital for this type of work,' said Dr. Fisher.

Dr. A. Radford, M.P. for Durban-Central, shared his colleagues' concern, especially at the 'grave shortage of medical personnel .... particularly in the Minister's own department'.

There was no immediate prospect of improving the shortage of trained men in the department or in private practice because of the paucity of training facilities. Only by subsidizing young men so that they would take up this profession and be under some obligation to go into mental service would the Minister relieve the position.

Nor did there seem to be any prospect of alleviating the shortage of nurses for the care of the mentally ill. The standard of entrance for nurses who want to take up psychiatric nursing had been lowered in an effort to encourage entrants. But this step had the opposite effect. Nurses who received general nursing training felt they were a little better than those who took up psychiatric training.

Experience in other countries had shown that the way to get nurses to take up psychiatric training was first to bring them into the general nursing field and allow them to receive their psychiatric training while learning to be nurses.

Dr. J. C. Jurgens, M.P. for Geduld, asked the Minister to extend the practice in existence in the Transvaal where the mental hospitals cooperated with outpatient clinics attached to the big hospitals.

The Minister, in reply, agreed that there should be outpatient departments for mental patients at the provincial hospitals as well as at the mental hospitals and informed the House that steps were already being taken to promote this idea. But there was the difficulty that the provincial hospitals did not fall under the jurisdiction of his department .... 'all we can do is try to persuade them'.

OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

TARIFF OF FEES FOR APPROVED MEDICAL AID SOCIETIES: SCHEDULE FOR PATHOLOGY

The fees for pathology have been omitted from the tariff book because of the frequent amendments and additions of new items which have to be made to the tariff on account of the advances in the speciality. The Transvaal pathologists' subgroup have already issued a tariff list, but delay has occurred with the issue of the tariff for the other centres. Members of the Association should in the meanwhile use the old tariff book and convert the fees to the decimal monetary system. Notice will be given when the tariff books are ready.

L. M. Marchand
Associate Secretary

28 Plaza Building
Bank Lane
Pretoria
23 February 1961

TARIEF VIR GOEDGEKEURDE MEDIESE HULPVERENIGINGS: GELDE VIR PATOLOGIE

Die gelde vir patologie is uit die tariefboek gelaat omdat daar met die ontwikkelinge in dié spesialiteit telkens wysigings en items vir nuwe toetse in die tarief aangebring moet word. Die Transvaalse afdeling van die groep patologie het alreeds 'n tarieflys uitgegee, maar daar het vertraging gekom met die uitgee van die tarief vir ander sentrums. Lede van die Vereniging moet dus solank nog die ou tarief gebruik en die gelde net omreken in die desimale muntstelsel. Kennis sal gegee word wanneer die tariefboeke gereed is.

L. M. Marchand
Medesekretaris

Plaza-gebou 28
Banksteeg
Pretoria
23 Februarie 1961

IN MEMORIAM

PATRICK ALBAN THORNTON, M.B., B.CH.

Mr. Lane Forsyth, of Cape Town, writes:

The recent death in East London of Dr. Patrick Alban Thornton, Medical Superintendent of the Emyanyana Leper Hospital, has removed from our midst a lovable and colourful character.

Dr. Thornton was born in Cape Town and received his education at St. Aidan's College, Grahamstown. He spent two years at Cape Town University before proceeding to Trinity College, Dublin. Here he came very much into his own — the Irish atmosphere struck a responsive chord in the nature of this young South African of Gaelic parentage. He learnt to love people and to enjoy life and living. He was bright and absorbed information with ease, and this capacity gave him ample time to pursue the sports which he enjoyed. He had already represented the University of Cape Town at soccer and cricket. He soon captained Dublin University at the same two games, and was awarded a coveted 'Trinity Pink'. He was elected to play for the 'Gentlemen of Ireland' team, and in 1926 won the cup for the best all-rounder in the country.

Dr. Thornton

After graduation he returned to the Union, and it was in keeping with his character that he should devote his professional life to the care of lepers.

He served in the South African Medical Corps during the Second World War in East Africa and in Egypt, and reached the rank of Lieutenant Colonel.

He will be remembered by his friends for his kindliness and geniality, and for the 'ditties' he used to sing in order to make a party 'go'.

I, however, will remember him as a true friend with whom I shared 'digs' for three years with great happiness. No stress or strain could rouse him to anger or provoke a harsh word from this loyal companion. So long, P.A.T.!

Our sympathies go out to his widow and his children.

JOHAN HENRY SYPKINS, M.B., CH.B. (EDIN.)

Mnr. V. H. Roodt van Nola, Transvaal, skryf:

As 'n leek, soms as 'n pasiënt, en altyd as 'n vriend van wyle dr. J. H. Sypkins van Barberton, wil ek graag 'n paar woorde van hulde bring aan sy nagedagtenis.

Dat sy onverwagte heengaan 'n skok was vir almal hier in die laeveld is seker, en dat sy afsterwe deur 'n groot aantal pasiënte betreur word, is ook seker.

Daar word vandag baie gesê en geskryf oor die geneesheer-pasiënt verhouding. Dit was juis in hierdie opsig dat dr. Sypkins uitstaande was. Hy was altyd vriendelik, belangstellend, simpatiek, en tog ook altyd heeltemal eerlik teenoor sy pasiënte.

Vir my as 'n leek is dit verstaanbaar dat 'n geneesheer soms in 'n moeilike geval 'n foutiewe diagnose kan maak. Wat egter baie belangrik is, is dat die geneesheer moet voortgaan om volle aandag aan die pasiënt te gee, om verder ondersoek in te stel, om behandeling toe te dien en om hom met hulp en raad by te staan.

So 'n geneesheer was dr. Sypkins. Hy was self uiters bekwaam, maar nogtans het hy nooit geaarsel om waar nodig 'n ander spesialis of geneesheer te raadpleeg nie. Hy was trouens 'n ware vriend en 'n beskermheer-geneesheer van sy pasiënte.

Aan sy weduwee en kinders wil ek graag my innige en opregte simpatie in hul verlies oordra.

PASSING EVENTS : IN DIE VERBYGAAN

Department of Anaesthesia, University of Cape Town. On Saturday 18 March a meeting will be held in the A-floor Lecture Theatre, Groote Schuur Hospital, Observatory, Cape, at 9.30 a.m. Dr. R. L. Kleinman will speak on 'Writing for journals'. All interested doctors are invited to attend this meeting.

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University of Cape Town and Association of Surgeons of South Africa (M.A.S.A.), Joint Lectures. The next lecture in this series will be held on Wednesday 15 March at 5.30 p.m. in the E-floor Lecture Theatre, Groote Schuur Hospital, Observatory, Cape. Prof. A. Kipps, Department of Bacteriology, University of Cape Town, will speak on 'Hospital infections'. All members of the Medical Association are welcome to attend this lecture.

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Mr. Barry Shandling and his wife and daughter, are at present in Toronto, Canada, where Mr. Shandling has been awarded a fellowship in surgery at the Hospital for Sick Children, Toronto, and is at present doing research on burns. With effect from July, 1961, Mr. Shandling has been appointed Associate Chief Resident at this hospital.

* * *

The National Occupational Safety Association is holding an Industrial Safety Convention at the Wanderers Club, Johannesburg, from 22 to 24 March 1961. All doctors who are interested are invited to attend this convention.

Hart-long Groep van die Universiteit van Stellenbosch. Die volgende vergadering van die Groep word gehou op 16 Maart 1961 in die Groot Voorlesingsaal, Kliniese-gebou, Karl Bremer-Hospitaal om 8 nm. Die program is soos volg:

1. Die Marfan-sindroom, Dr. P. W. A. Botha.
2. Kort kliniese mededelings. (Lede word versoek om interessante EKG's, röntgenfoto's, ens. saam te bring vir bespreking.

Alle belangstellendes word vriendelik uitgenooi om die vergadering by te woon.

* * *

Research Forum, University of Cape Town. The next meeting will be held on Thursday 16 March at 4 p.m. in the Tutorial Room of the Pathology Department, Medical School, Observatory, Cape. Dr. P. Smythe will speak on 'The treatment of tetanus neonatorum with intermittent positive-pressure respiration'. This will be followed by a Staff Clinical Conference held in the Falconer Lecture Theatre, Groote Schuur Hospital, Observatory, Cape, at 5.15 p.m. All general practitioners and others who may be interested are invited to attend these meetings.

* * *

South African Institute for Medical Research, Johannesburg, Staff Scientific Meeting. The next meeting will be held on Monday 20 March at 5.10 p.m. in the Institute Lecture Theatre. Mrs. Diedrich and Mrs. Gilich will discuss 'The use of the autoanalysis in biochemical examinations'.

Members are reminded that they should notify any change of address to the Secretary of the Medical Association of South Africa at P.O. Box 643, Cape Town, as well as to the Registrar of the South African Medical and Dental Council, P.O. Box 205, Pretoria. Failure to advise the Association will result in non-delivery of the *Journal*. This applies to members proceeding overseas as well as those who change their addresses within the Union.

* * *

Eastern Transvaal Branch (M.A.S.A.) Jooste Cup Golf Competition. Entries are invited from all members of the Medical Association for the 'Jooste Cup' Floating Trophy and many handsome subsidiary prizes, to be played at the Reading Country Club, New Redruth, Alberton, Transvaal, over 18 holes on Sunday 26 March 1961, starting at 12 noon. Entries may be submitted to Dr. W. Sacks, Evelyn Mansions, Third Street, Springs, by Tuesday 21 March 1961.

Tak Oos-Transvaal (M.V.S.A.) 'Jooste Cup' Gholfwedstryd. Lede van die Mediese Vereniging word versoek om aan die Jooste Cup' Wisseltrofee en ander aantreklike aanvullende pryse deel te neem wat oor 18 putjies gespeel sal word te Reading Country Club, New Redruth, Alberton, Transvaal, op Sondag 26 Maart 1961, om 12 middag. Inskrywings moet aan Dr. W. Sacks, Evelyn Mansions, Derdestraat, Springs, gestuur word om hom teen Dinsdag 21 Maart te bereik.

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Dr. M. M. de la Harpe, M.Med. (O. et G.) (Pret.) M.R.C.O.G., het op 1 Maart 1961 begin om privaat te praktiseer as Vroue-arts en Verloskundige te Mediese Sentrum 412, Pretoriusstraat 319, Pretoria. Telefone: Spreekkamer 20319, woning 782920.

Dr. M. M. de la Harpe, M.Med. (O. et G.) (Pret.) M.R.C.O.G., commenced private practice as a Gynaecologist and Obstetrician on 1 March 1961, at 412 Medical Centre, 319 Pretorius Street, Pretoria. Telephones: Rooms 20319, residence 782920.

* * *

Victoria Hospital, Wynberg. A meeting will be held at the Nurses' Home, Victoria Hospital, Wynberg, on Tuesday 21 March at 8.15 p.m. Dr. J. Heselson will discuss 'Recent advances in vascular surgery' and Dr. M. J. Bailey will speak on 'Some aspects of hypertension'. All practitioners are cordially invited to attend this meeting.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

WESTPROD INTERNAL DEFIBRILLATOR — PACEMAKER

Westdene Products (Pty.) Ltd. announce the introduction of the Westprod Internal Defibrillator — Pacemaker, manufactured by E. J. Middleton (Electronics) (Pty.) Ltd. of Johannesburg, and supply the following information:

This is a combined unit consisting of a defibrillator and a pacemaker. Both units operate off 220/250 volt 50 cycle A.C. The Defibrillator output to the patient is 50 - 300 volts. The impulse rate is 0·15 seconds in duration and a non-locking switch allows impulses to be repeated as required. A safety automatic relay switch in the patient's circuit affords complete protection. A neon lamp indicates the impulses.

There is a selector switch to change from defibrillator to pacemaker. The Pacemaker voltage to the patient is 0 - 50 volts and the pulse rate to the patient is 35 - 130 per minute. The pulse rate indicator is a neon lamp flasher and is the common power indicator. Both are complete with electrodes and cardiac wire.

Further information may be obtained from Westdene Products (Pty.) Ltd., P.O. Box 7710, Johannesburg.

BOOK REVIEWS : BOEKBESPREKINGS

USE OF RADIATION AND ATOMIC ENERGY

Radiation Hygiene Handbook. A practical reference covering the industrial, medical, and research uses of radiation and atomic energy with special applications to the fields of health physics, industrial hygiene, and sanitary engineering. 1st edition. Editor-in-Chief: Hanson Blatz. In 23 sections. $27.50. New York, Toronto, London: McGraw-Hill Book Company, Inc. 1959.

The title of the volume does not do justice to the comprehensive nature of this reference book on the industrial, medical and research uses of radiation and atomic energy.

The book is divided into 23 sections, each section compiled and edited by one or more scientists who are leaders in their particular field and attached to leading institutions in the USA.

Each section comprises an up-to-date reference and data source on the use of radiation energy and especially atomic energy, in health physics, industrial hygiene and sanitary engineering.

The individual chapters provide very complete though condensed information on the particular subjects; as an example one may quote the chapter on 'Radiation detectors and measurements', which is covered by 6 authorities and includes full descriptions of ionization chambers, scintillation counters, proportional and geiger counters, photographic monitoring of radiation, chemical monitoring and dosimetry using the optical properties of solids.

The unorthodox way of numbering the pages by preceding each page number with that of its section, and commencing each section at page 1, greatly facilitates the orientation of any page in relation to its contents. The same system applies to diagrams.

This volume is therefore a welcome addition to the literature on radiation energy. It does not supplant any of the standard publications in this field, but constitutes an indispensable reference book in any department using ionizing radiations, more especially radio-active isotopes.

J.M.v.N.

DISEASES OF THE ANUS AND RECTUM

Synopsis of Treatment of Anorectal Diseases. By Stuart T. Toss, M.D., F.A.C.S., F.I.C.S. Pp. 240. 59 illustrations. South African price: R5.52½. Local agents: P. B. Mayer, P.O. Box 713, Cape Town, and Westdene Products (Pty.) Ltd., Medical Book Dept., Box 7710, Johannesburg. St. Louis: C. V. Mosby Co. 1959.

This excellent little book contains a wealth of information clearly set out and easily accessible.

It contains 27 chapters, 25 of which are short, fairly complete descriptions of anorectal conditions ranging from constipation and impaction to incontinence, foreign bodies and trauma.

The descriptions of the more common conditions are detailed and informative. It is to be recommended to students and practitioners who will find it a most useful book.

This small book is excellently bound and is well illustrated. It has no bibliography, however, and for its size is relatively expensive. P.A.T.

LEUKOCYTE ANTIGENS AND HUMORAL ANTIBODIES

Leukocyte Antigens and Antibodies. By Roy L. Walford, M.D. Pp. 182+ix. $6.75. New York and London: Grune & Stratton, Inc. 1960.

The author has provided a timely review of a rapidly expanding field. His book is mainly concerned with leukocyte antigens and humoral antibodies directed against them. The rôle of the leukocyte in delayed hypersensitivity and its possible rôle in antibody transport and synthesis are not considered.

Techniques that may be employed are fully covered. These include the various ways of obtaining cell suspensions, and of cell fractionation to separate nuclei, mitochondria, granules, microsomes, or the soluble fraction of cells.

The relative merits of the techniques used in the detection and study of leukocyte antigens and antibodies are assessed. A particularly important advance has been the development of the antiglobulin consumption test.

Useful discussions are included in separate chapters on drug-induced neutropenia and agranulocytosis; the LE factor; leukocyte antibodies in relation to the homograft reaction; and, of course, on the clinical significance of leukocyte antibodies.

This book fills a real need in assembling and carefully assessing the available information, and should be of interest both to the clinician and the laboratory worker. W.B.

CORRESPONDENCE : BRIEWERUBRIEK

INCOME CEILING £2,300

To the Editor: At a special meeting of the Cape Western Branch of the Medical Association held recently, the consensus of opinion was that the £2,300 (R4,600) *per annum* income ceilling, agreed to by Federal Council as a basis of negotiation with SANSOM, was unrealistic, mainly because it did not take into account the number of dependents, nor was there any means of proving the income figure as correct.

After the meeting I gave the matter a great deal of thought and have evolved a basis which I feel is as realistic as possible, with the proof in black and white available to the insurance company when accepting membership and to Federal Council in the event of any dispute, viz the member's average income tax assessment taken over, say, a period of three years. The figure I suggest is a ceiling assessment of £150 (R300) *per annum*, which of course includes the income, if any, of the member's wife. A single man earning £2,300 *p.a.* pays £270 tax. A married man with two children, earning £2,300 *p.a.* pays £162 tax. A single man earning £1,500 *p.a.* pays £158 tax. By accepting a tax assessment ceiling of £150, the member who will be entitled to preferential rates will earn just below £1,500 *p.a.* if single, and just below £2,300 *p.a.* if married with two dependents.

The receiver of revenue applies the sliding scale which we require to adjust the earnings of the family-man realistically, and I think we can accept that the assessment will be correct in the vast majority of cases. Any member who does not wish to produce his tax assessment will be regarded as being above the ceiling level and will not qualify for preferential tariff. I may add that anyone who is clever enough to get away with anything from the receiver of revenue will be far too clever for us mere doctors, and I will be quite prepared to accept this figure without any qualms.

I see no reason why this basis should not be used universally in dealing with medical aid societies, insurance companies, and with the Medical Services Plan.

B. W. Myers

4 Orange Street
Cape Town
23 February 1961

ALTAFUR AND ALCOHOL

To the Editor: The manufacturers of 'altafur' have recently warned doctors that persons taking altafur should avoid alcohol for 1 week. Before this warning, the following manifestations were seen in a patient who was treated with altafur; they were sufficiently alarming to cause considerable anxiety to the patient and to myself.

The patient, a healthy young male subject, was given altafur, 1 tablet *q.i.d.*, for a staphylococcal urethritis, in which the organism was found to be sensitive to altafur.

Four days after treatment commenced, I received an emergency call to see the patient who complained of a severe constriction across his chest, together with a marked feeling of heat and flushing, especially behind his ears, and also a marked 'thumping of his heart against his chest'. On examination his face was markedly flushed, his pulse rate was 100/min. and he appeared to be in some distress, mental perhaps more than physical. His appearance was similar to that seen in a person taking alcohol after antabuse, and I in fact told him 'somebody has been doctoring your drinks'. He then stated that he did not drink, but when my assertion was repeated, he suddenly remembered that he did drink Export Ale the previous afternoon, and had experienced similar symptoms, though to a lesser degree. During the second afternoon, he took a tablet at 4 p.m., and at 4.30 p.m. he again consumed 2 bottles of Export Ale. The symptoms appeared at 4.40 p.m. and were particularly frightening during the next 30 minutes. When I saw him he had already started to improve, and by the next morning he felt quite normal again.

The efficacy of altafur in cases of staphylococcal infection appears to be undoubted, but it should be strongly impressed on all patients taking altafur that alcohol, even in small quantities, can produce symptoms that are, to say the very least, alarming.

C. K. Edelstein

District Surgeon
Wynberg
Cape Town
20 February 1961

FEEDING PROBLEMS OF YOUNG CHILDREN

To the Editor: I should like to express my appreciation of the excellent article on 'Feeding problems of young children of pre-school age' which Dr. Mirvish contributed to the *Journal* last week.[1]

Having dealt with similar problems in cooperation with paediatric specialists and general practitioners for over thirty years, I consider it one of the finest and most helpful contributions I have seen — and I have read many. If all doctors could deal with the problems of feeding from the earliest period in this practical, psychological and human way, then many of these and other later behaviour problems would not arise.

'Then surely would good digestion wait on appetite and health on both' — as Shakespeare tells us.

Mary Mackie

Fairfield Hotel
Wynberg
Cape Town
20 February 1961

1. Mirvish, I. (1961): S. Afr. Med. J., **35**, 121.